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OPERATIVE TREATMENT OF RETINAL DETACHMENT WITH ELECTROCOAGULATION

ARNOLD KNAPP, M.D

NEW YORK

It is now being generally accepted that the principal factor in the operations for detachment of the retina is the closure of the hole in the retina which allowed the escape of fluid from the broken-down vitreous into the subretinal space. Thus the contention of de Wecker in 1870, and of Leber in 1882, that the retinal tear was the direct cause of the detachment is now confirmed. According to Vogt,[1] Galezowski, in 1903, was the first to treat systematically the retinal tear with ignipuncture, after aspirating the subretinal fluid, but the method did not gain recognition until it was rediscovered and worked out adequately by Gonin in 1923. General trial of the operation of ignipuncture soon led to a recognition of its disadvantages, consisting, in addition to its limited field of application, of loss of vitreous, destruction of retinal tissue and late hemorrhage. In the search for less dangerous procedures, Lindner and Guist suggested cauterization with potassium hydroxide, U. S. P. With this method the results were definitely better, but the multiple trephinings are most tedious and difficult, and the possibility of perforation of the choroid and intra-ocular hemorrhage is a real objection. Then, in 1930, treatment of retinal detachment with diathermia was advocated by Weve, Larsson and Safar; this is the method which is the most popular at present, and it offers possibilities of great promise. The purpose of these methods was to produce an adhesive choroiditis by which the retinal hole is shut off. Ignipuncture, particularly with the Paqualin cautery, may cause too great an effect, and by destroying very delicate tissues like the retina and vitreous irremediable damage is done. According to Stein,[2] there is no physiochemical difference between electrocoagulation and cauterization with potassium hydroxide; these methods are undoubtedly much less severe and more reliable than ignipuncture.

Read before the American Ophthalmological Society, Washington, D. C., May 10, 1933.

1. Vogt, Alfred: Operative Treatment of Detachment of the Retina: Abstract of the Official Review Presented Before the International Ophthlamological Congress, April 18, 1933.

2. Stein: Klin. Monatsbl. f. Augenh. **90**:551, 1933.

In diathermia, the high frequency current is used to produce coagulation of the tissues. This is accomplished by the application of needle electrodes to the eyeball at the site of the tear. With careful dosage the needle perforates the sclera and the choroid, and a circumscribed area of the choroid is coagulated. The subretinal fluid then escapes through the punctured hole, and the retina becomes attached to the coagulated choroid and, in favorable cases, adheres. The vitreous is shut off from the subretinal fluid by the seclusion of the hole, that is, by the adhesion of the retina to the choroid in an area surrounding the tear. The needle electrodes vary in size and thickness. Weve uses long, thin needles with which he tries to puncture the retina at the margin of the tear; with the ophthalmoscope he observes the effect of his operation. Safar's needles are only 1.5 mm. in length in order to prevent too deep penetration, as he fears injury to the retina and the vitreous; Safar's needles also come in series of three, or collections of six or seven of the so-called brush electrodes, which are attached to an insulated stalk and are in the form of short stick pins which can be left in situ to prevent too early escape of the subretinal fluid. The needle punctures are made about 2 mm. apart, and their number depends on the retinal condition that is present and the object to be achieved. Adhesive choroiditis can be produced with a mild current. A current of low intensity of not more than 30 ma. to each needle is used; the needle perforates the sclera quickly and without force. As soon as the perforation takes place, the needle is promptly withdrawn or the current is shut off and the needle is left in situ. The proper dose is extremely important. With a too long or a too strong dose the coagulated tissues continue to be heated and approach burning, whereby certain advantages of coagulation such as a soft scar are lost. Too heavy doses damage the retina and produce opacities of the vitreous and injury to the macula. The older the retinal detachment the more friable is the retina and the greater is the danger of secondary tears following the operation. While hemorrhages in the vitreous also occur with diathermia, they are much less frequent than with ignipuncture. After diathermia, white coagulated areas are seen with the ophthalmoscope; then grayish-white dotted cloudy areas with a grayish reflex like a fresh choroiditis are noted; gradually, after two or three weeks, the individual puncture areas appear, pigment develops and the picture of an old choroiditis is presented.

Proper indications for operation must be present. The most important indication is that the retina should be capable of being unraveled or replaced; it is also essential that no bands or folds be present and that the retina should not be shrunken; in other words, that none of the changes in the detached retina which come with age should have occurred.

The color of the detached retina gives valuable information. The vitreous must not be disorganized and the tension of the eyeball not markedly reduced. This holds true particularly for aphacic cases. An important step is the examination for holes and tears and the determination of their number and position. The principles laid down by Gonin [3] and Amsler [4] will be found of great value. This examination may require considerable time, but the discovery of the tear and its localization often mark the difference between success and failure. In cases in which no hole in the retina is found, operations have nevertheless been performed, and with some, although a smaller percentage of, success. In these cases the presumptive site of the beginning of the detachment is attacked.

After one year in which I used ignipuncture (twenty cases), and a second year in which I employed cauterizations with potassium hydroxide (nine cases), I am now trying the diathermia treatment, and during the past year I have operated in twelve cases which are the subject of this communication.

The operative technic which I have followed is the one which Safar described carefully in his recent monograph.[5]

REPORT OF CASES

CASE 1.—E. G., aged 28, seen on Sept. 6, 1932, stated that her left eye became affected six weeks before. Vision in the left eye was 6/36. The field showed a defect in the upper third. Fine opacities were seen in the vitreous. There was a detachment of the retina in the lower half with a tear at the ora between 5 and 6 o'clock. On September 13, a line of coagulations was made 15 mm. from the limbus to enclose the tear. Subsequently the coagulations were found to have been placed too far temporally, leaving a nasal gap which led to a detached area of the retina. Temporary tenotomy of the inferior rectus muscle was therefore done on October 6. After proceeding from the coagulated spots of the first operation, new coagulations were placed nasally. This procedure closed the hole; the retina was replaced, and on April 5, 1933, the vision in the left eye was 6/9—. The field was normal.

CASE 2.—F. T. R., aged 26, was referred by Dr. S. B. Marlow, of Syracuse, N. Y., with a history of detachment of the retina in the right eye since June. On Sept. 14, 1932, the vision in the right eye with —7 sph. —1.25 cyl. horizontal was 6/12. The field showed a defect up and in. The retina presented a shallow detachment in the external and lower fields, with five holes at 9:30, 7:30, 6:45 and 6 o'clock. On September 16, electrocoagulation was done. The lower and outer periphery of the sclera was exposed; tenotomy of the inferior rectus muscle

3. Gonin, J.: Ann. d'ocul. **168**:689, 1931.

4. Amsler, M.: Klin. Monatsbl. f. Augenh. **86**:1, 1931.

5. Safar: Behandlung der Netzhautabhebung mit multipler diathermischer Stichelung, Berlin, S. Karger, 1933.

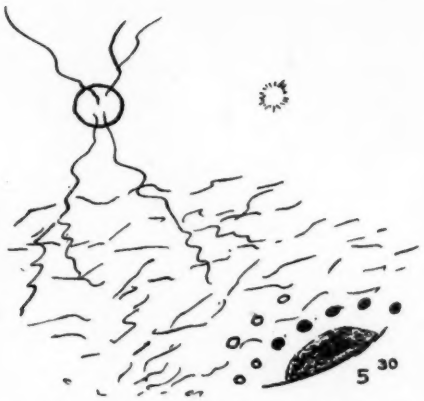

Fig. 1 (case 1).—Defect in the upper third of the field. Vision in this eye (left) was 20/100. On Sept. 9, 1932, a line of electrocoagulations was made 15 mm. from the limbus at 5:30 o'clock. On October 6, a second operation was performed. On April 5, 1933, vision in the left eye was 20/30 and the field was normal.

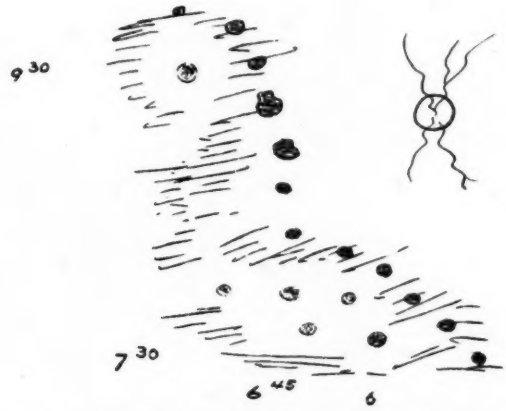

Fig. 2 (case 2).—Defect in the field, up and in. Vision in this eye (right) with —7 sph. —1 cyl. was 20/40. On Sept. 16, 1930, electrocoagulation was done at 20 mm. from 10 to 6 o'clock. On April 14, 1933, vision in the right eye with glasses was 20/30.

Fig. 3 (case 3).—Contraction of the field to a horizontal slit. Vision in this eye (right) with a +4 sph. was 5/200. On Oct. 20, 1932, eight punctures were made at 15 mm. On April 4, 1933, vision in the right eye with glasses was 20/40. The field showed a peripheral defect, down and out.

was performed. Twelve coagulations were made in a curved line 20 mm. from the limbus from 10 to 6 o'clock. On March 24, 1933, the vision in the right eye with glasses was 6/9; the retina was reattached and the field was normal.

CASE 3.—L. F. S., aged 69, was referred by Dr. W. S. Atkinson, of Watertown, N. Y., on Oct. 12, 1932. The patient stated that the vision in the right eye failed two months before. Vision in that eye was 1/60. The field was contracted to a horizontal area nasally from the point of fixation. There was a shallow detachment of the retina below, with an area of rarefied retina with many fine holes in the upper and nasal periphery at 1:30 o'clock. On October 20, electrocoagulation was done. The sclera was exposed up and in between the superior and external rectus muscles, and at 15 mm. eight punctures were made. On March 24, 1933, the retina was in place. Vision in the right eye was 6/12. The field was normal.

CASE 4.—L. M., aged 13, referred by Dr. W. A. Holden, New York, was seen on Sept. 9, 1932, with an indefinite history of failing sight in the right eye for three months. Vision in the right eye with —3.5 cyl., axis 75° was 6/60. The field was defective in its temporal half and there was a paracentral scotoma; the retina showed a general shallow detachment. No hole could be found. The patient stated that the failure of vision seemed to begin with a blur to the outer side and below. Operation was performed on November 4. The inner half of the sclera, 12 mm. back from the limbus, was exposed and the internal rectus muscle tenotomized. Fourteen coagulations were applied in an arc passing from 1 to 5 o'clock. On December 9, vision in the right eye was 6/24. There was a peripheral defect in the temporal field but no scotoma. Examination on April 1, 1933, showed that the sight had recently become worse and was reduced to 6/36. There was a shallow detachment nasally and also above. Another operation is planned.

CASE 5.—E. R., aged 40, was seen on Nov. 17, 1931, as the sight in her left eye had failed three days before. Vision in the left eye with —4 sph. —3 cyl. h. was 2/60. The field showed a large defect in the upper and nasal parts. There was a large cystic detachmment of the retina down and out, but no hole could be seen. The process seemed to be choroidal and probably tuberculous. The patient reacted to tuberculin and for three months was given tuberculin systematically, at first with improvement; later the detachment approached the macular region, and an operation was decided on. On Feb. 25, 1932, a Guist operation was performed. Ten trephine openings were applied 15 mm. from the limbus in the lower sclera. The vision improved to 6/60. The field showed a small defect above. A shallow detachment remained central to the cauterized areas, and in the nasal periphery there was a deep retinal hemorrhage. Treatment with tuberculin was continued, and on May 4 vision in the right eye was 6/14. In November, 1932, the detachment had recurred nasally; the field defect again approached the center with reduction of vision to 6/60. On December 8, electrocoagulation was done. The sclera was exposed below; the old cauterized areas were easily recognized. Ten electrode points were applied 20 mm. distant from the limbus, and three electrodes were placed at 10 mm. in the lower sclera. This procedure was followed by considerable reaction, and a large subretinal hemorrhage occurred. The retina remained adherent, and the fundus gradually became clearer except for dense black vitreous (hemorrhage) opacities. The administration of tuberculin was continued. On April 21, 1933, vision in the left eye was 6/24. There was a shallow detachment below, with

some blood in the vitreous and in the retina. The field showed a small defect in the upper periphery.

CASE 6.—M. C., aged 35, stated that for two weeks vision in the right eye had been affected. On May 1, 1932, vision in the right eye was 6/60. The field showed a defect in the upper half. There was a detachment of the retina in the lower half, including the macula. A tear at the ora extending from 6 to 7 o'clock was found. On May 17, a Guist operation was performed. Six trephine openings were made 16 mm. distant from the limbus, and potassium hydroxide was applied. The retina did not become reattached. Vision in the right eye was 6/36. There was a detachment of the retina in the lower half including the macula, as before. On December 10, electrocoagulation was done. The lower part of the sclera was again exposed but with difficulty because of adhesions from the previous operation. Fourteen electrode points were applied 18 mm. distant from the limbus, and several single ones more anteriorly. The retina became reattached. The line through the macula remained. On April 21, 1933, vision in the right eye was 6/9. The field showed a slight peripheral defect above.

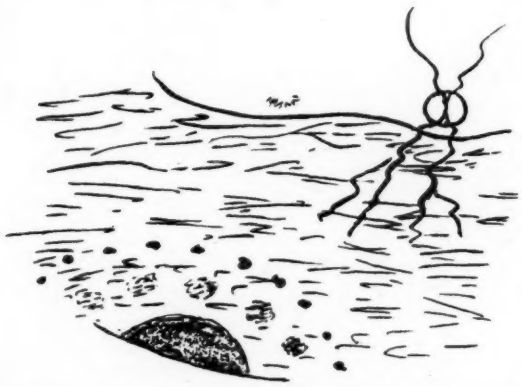

Fig. 4 (case 6).—Defect in the upper half of the field. Vision in this eye (right) was 20/200. On May 17, 1932, a Guist operation was performed, with six trephine openings at 16 mm.; on December 10, electrocoagulation. On April 21, 1933, vision in the right eye was 20/30, with a peripheral defect in the field.

CASE 7.—A. H., aged 50, was seen on Feb. 16, 1932. The trouble in his right eye began with a hemorrhage in the vitreous. Vision was reduced to ability to see the fingers at 0.6 meter. Later a detachment of the retina could be observed in the outer field, and a triangular tear at 3 o'clock was seen. A Guist operation was performed on March 31. Several trephine openings were made temporarily from 17 to 20 mm. distant from the limbus to enclose the tear, and potasium hydroxide was applied. On April 11, vision in the right eye was 6/60. The field showed a defect down and in. Below, a fibrinous exudate was seen in the vitreous. In the temporal fundus pigment with deep hemorrhage could be seen. Vision then improved to 6/14, and the area surrounding the tear seemed to be adherent, but there was a detachment above. The eye then became worse, and on December 12, vision in the right eye was 1/60. The field was concentrically contracted. There was a general detachment of the retina with the old tear visible at 8 o'clock. On December 14, electrocoagulation was done. After tenotomy of the external rectus muscle the sclera was exposed temporally. At 18 mm. back of the limbus the seven-pronged curved brush electrode was applied posterior to the insertion of the

inferior oblique muscle, and considerable fluid vitreous escaped, so that the eye collapsed and no further punctures could be made. There was no result, and on January 13, vision in the right eye was 2/60. A general detachment was present.

CASE 8.—H. E., aged 25, seen on Jan. 9, 1933, stated that the sight in the left eye had been poor for two months. Vision in the left eye with —7 sph. was 6/60. The field showed a defect down and in. A large detachment was found in the upper and outer fundus with two tears at 1 : 30 o'clock. On Jan. 14, 1933, electro-

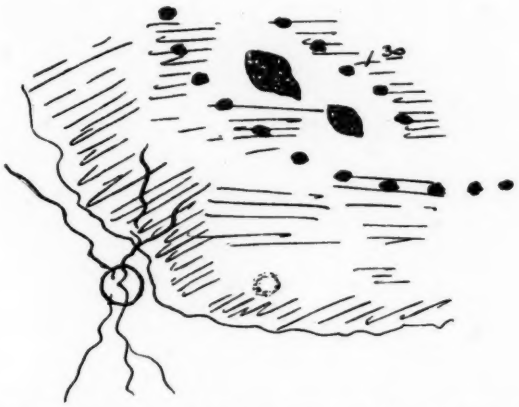

Fig. 5 (case 8).—Defect in the field, down and in. Vision in this eye (left) with —8 sph. was 20/100. On Jan. 14, 1933, electrocoagulation was done at 16 mm. On April 19, vision in the left eye with glasses was 20/70. The field showed a slight peripheral defect.

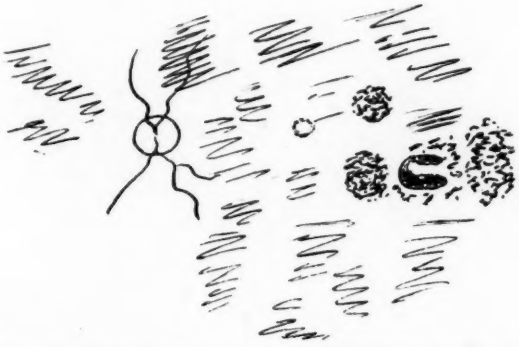

Fig. 6 (case 9).—Field preserved only in the upper and outer quadrant. Vision in this eye (left) with —5.5 sph. —2.5 cyl. was 1/60. On Jan. 25, 1933, electrocoagulation was done, a seven-pronged electrode being applied at 24 mm. On March 8, electrocoagulation was again performed, at 20 and 24 mm. Vision in the left eye was 20/100. There were macular changes and a slight nasal defect in the field.

coagulation was done. The sclera was exposed up and out; nine electrodes were then inserted 16 mm. distant from the limbus between 12 to 3 o'clock, and five electrodes 14 mm. back from the limbus; at each periphery a single electrode was again introduced without current to allow the subretinal fluid to escape. On April 15, vision in the left eye was 6/24. The field showed a slight defect in the lower and outer periphery. The retina was in position.

CASE 9.—A. M., aged 60, stated on Jan. 4, 1933, that the sight in her left eye was lost about two months previously. Vision in the left eye with —5.5 sph. —2.5 cyl.h. was 1/60. The field showed a nasal defect. There was a general detachment in the outer half of the fundus with a tear at 4 o'clock. On January 25, electrocoagulation was done. After tenotomy of the external rectus muscle the temporal surface of the sclera was exposed, showing the insertion of the inferior oblique muscle. The curved seven-pronged brush electrode was applied 24 mm. from the limbus; this was followed by constant oozing of the vitreous. The ophthalmoscope showed that the coagulated area was too high, and another attempt was made to make a puncture below. This was unsuccessful, as the eyeball had collapsed. Another operation was performed on March 8. The preparatory dissection was difficult on account of the presence of adhesions and hemorrhage. Twenty millimeters back and below the horizontal meridian the seven-pronged electrode was applied, and in addition, a number of single applications were made. This was followed by considerable reaction which lasted for several days. On April 28, the retina was found adherent. Vision in the left eye was 6/36. There were slight

Fig. 7 (case 10).—Defect in the upper half of the field. Vision in this eye (right) was 20/200. On April 11, 1933, electrocoagulation was done at 18 mm. Examination on May 1 showed vision of 20/200 in the right eye, a macular lesion and detachment, and a peripheral defect of the field, up and in. A second electrocoagulation was done on May 18 at 13 mm. On June 8, the retina was reattached, vision in the right eye was 20/200, and macular changes were noted.

macular changes. The hole could not be recognized in the pigmented choroidal area. The field showed slight nasal contraction.

CASE 10.—J. A. B., aged 37, was referred to me by Dr. G. E. Dailey, of Gloversville, N. Y., on April 5, 1933, with a history of loss of sight in the right eye for six weeks. Vision in the right eye was 6/60. The field showed a defect in the upper half. There was a detachment of the retina in the lower half, with a large semicircular tear at the ora between 6 and 8 o'clock. The detachment extended upward and included the macular region. On April 11, electrocoagulation was done. The sclera was exposed down and out, and 18 mm. distant from the limbus fourteen electrodes were applied to circumscribe the tear at the ora. On May 1, vision in the right eye was 6/60. The field showed a peripheral defect up and in. The tear was surrounded by a whitish-red coagulated area except for a gap from which a shallow detachment began and extended nasally. The macular region showed a shallow detachment. On May 18, 1933, a second electrocoagulation was done. The sclera was exposed below after tenotomy of the inferior rectus

muscle. Thirteen millimeters back from the limbus nine electrodes were applied in an area next to the old coagulated points extending nasally and forward so as to shut off the nasal gap. On June 8, vision in the right eye was 6/60. The retina was reattached, the peripheral field was normal and there were faint macular changes.

CASE 11.—M. J. C. came to see me on April 11, 1933, stating that the sight in his left eye had failed two weeks before. Vision in the left eye with —6.50 sph.

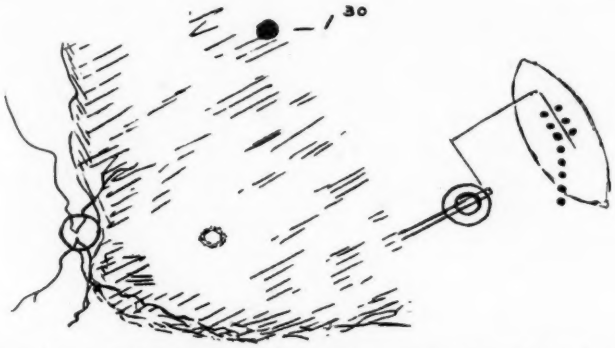

Fig. 8 (case 11).—Defect in the nasal and upper portion of the field. Vision in this eye (left) with —1.5 sph. was 3/200. On April 14, 1933, electrocoagulation was done at 17 mm. On June 22, vision in the left eye with —6.5 sph. —0.75 cyl. was 20/30—. The retina was in place and the field normal.

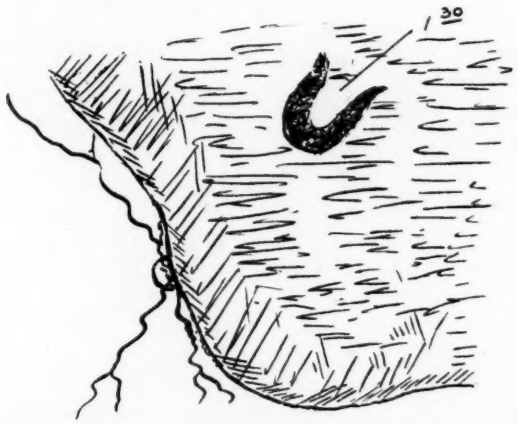

Fig 9 (case 12).—Field preserved in only the upper and inner quadrant. Vision in this eye (left) amounted to perception of fingers at 0.5 meter. On April 14, 1933, electrocoagulation was done, a seven-pronged electrode being applied at 18 and 22 mm. On June 2, vision in the left eye with —16 sph. was 20/100. The retina was in place and the field normal. There were some opacities in the vitreous and macular changes.

enabled the patient to see fingers at 0.5 meter. The field showed an upper and nasal defect. The fundus showed extensive detachment in the temporal half limited below by branching subretinal lines. A careful search revealed only a small hole at 1:30 o'clock. On April 14, electrocoagulation was done. The sclera was exposed up and out. A six-pronged brush electrode was applied 17 mm. back from the limbus, and some vitreous escaped; five single coagulations were then

made in a line, directly downward. Several openings were dilated, but no further escape of vitreous occurred. On May 4, vision in the left eye with —6.5 sph. — 0.75 cyl. h. was 6/9 —. The field showed a slight peripheral defect down and in. The retina was in place.

CASE 12.—B. B., aged 36, was seen on April 22, 1933, with the history that the sight in his left eye had failed two weeks before. In the left eye a — 16 sph. enabled the patient to see fingers at 0.5 meter. The field was preserved in only the upper and inner quadrant. There was a large balloon-shaped detachment in the upper and outer part of the fundus with an enormous horseshoe-shaped tear base up and out at 1:30 o'clock. On April 25, electrocoagulation was done. After exposure of the sclera in the upper and outer area in the 1:15 o'clock meridian, the seven-pronged electrode was applied 18 mm. back, and watery fluid escaped. The ophthalmoscope showed that the meridian was correct, but that the coagulated area was not far enough back from the limbus. Three additional punctures were then made 22 mm. back from the limbus together with many single ones. An attempt was made to aspirate the fluid but without success. At the end of the operation there was a good pink reflex in the upper and outer fields of the fundus. Considerable reaction in the form of swelling, redness and pain followed this operation, and lasted one week. On June 6, vision in the left eye with — 16 sph. was 6/36. The field was normal and the retina in place. There were old opacities in the vitreous and myopic macular changes.

SUMMARY

The results in these cases are shown in the accompanying table.

Results in Twelve Cases of Retinal Detachment

	Recovered	Improved	Not Improved
Tear at the ora	3	0	..
Holes	5	1	1
No holes	..	1	1
	8	2	2

It is, of course, too soon to say that these results are final, and I hope to make a supplementary report, I feel confident that this will prove to be the most successful operation for the cure of retinal detachment that yet has been advanced. The technic is simple, and the procedure seems to be harmless to the eyeball. I have used the Safar electrodes with satisfaction, and the principle on which they are constructed is ingenious and efficient. Of course, as in all operations for retinal detachment, preliminary study of the case is most important, and repeated examinations may be necessary before a plan of operation can be decided on. Exact localization of the detachment is desirable, and this requires painstaking care and practice. A shallow detachment with a peripheral tear offers no difficulties. An area large enough to include the hole can readily be coagulated. If the hole is situated far back, the operation becomes more difficult, as the area must be struck with the curved brush electrodes. In large balloon-like detachments the exact localization is impossible unless, as Safar suggests, the subretinal fluid is first

allowed to escape, and when the retina is replaced, an accurate localization is possible and the electrocoagulation can be done. In cases in which no hole is visible the prognosis is uncertain; the only procedure seems to be the coagulation of a large area at the point where the detachment began. Inspection of the fundus during the operation with the aid of a contact glass as Weve suggests is valuable. The possibility of cataract developing from coagulation in the area of the ora serrata must be considered as a complication.

Finally, it must be remembered that the operation for retinal detachment is only a mechanical relief of a symptom—a symptomatic operation—and that there is still much to be learned regarding the pathology of the retinal changes in detachment and the relation of the vitreous to the detached retina. The favorable results that have been obtained by operations in a condition which was previously considered hopeless are bound to stimulate study, and important additions to the knowledge of retinal detachment should follow.

ABSTRACT OF DISCUSSION

DR. PETER C. KRONFELD: I personally believe that the outcome of the operation for retinal detachment does not depend on whether one utilizes the Guist method or electrocoagulation. I believe that my statistics at this time, especially in a small series of fifteen or twenty cases, show much more variation in types of detachment and their response to the operation. In my experience I think of one mistake I have made, namely, the failure to provide for proper drainage of the subretinal space. In the Guist operation there are trephine holes through which the subretinal fluids can drain a few days after the operation. The small holes made with the diathermy needle are closed almost immediately. I have come to the conclusion that it is necessary to make sure at the end of the operation that the subretinal space is emptied, because it can be readily seen that in order to get good adhesion the retina must be in contact with the choroid and remain that way for a few days. In the last few cases I have therefore combined the electrocoagulation method with one or two trephine holes, after the method of Guist, in order to obtain good drainage of the subretinal space.

DR. WALTER B. LANCASTER: My experience in condemning ignipuncture agrees with that of Dr. Knapp. I had used the Guist operation, but it seemed to me that a little different method of cauterization certainly could be devised. The objection to trephining, and this applies to what Dr. Kronfeld said, is the possibility of hemorrhage. Occasionally a hemorrhage occurs when the coagulation needle is used, but it can be stopped instantly. In fact, that is the method used by general surgeons in stopping hemorrhage, for instance, of the brain.

As to the length of time to keep the electrocoagulation current on, Dr. Knapp says that as soon as he has punctured he withdraws the needle. I have left the current on longer than that, perhaps because I use a weak current. I do not like brush electrodes. When the current is applied at so many points it is necessary to use a stronger current, and the result is more reaction and less control. I use the single or double points, possibly treble, but I prefer the single or double points. I do not place the punctures so near together as Dr. Knapp advised. On the other hand, I cover a larger area.

DR. LUTHER C. PETER: As Dr. Knapp has pointed out, among the advantages of this method is that in the hands of those who do little work of this kind electrocoagulation offers the best results. Furthermore, one of the advantages of this method lies in the fact that one can cover a much larger surgical area, and that to my mind is important also. It does not require so many reoperations. If the surgical field goes far enough beyond the immediate area of the tear, or in the area of the ora serrata, one goes well beyond the area of detachment and the results in my experience are much better.

DR. MARK J. SCHOENBERG: Of course there are two steps in handling a case of detachment of the retina. One is the diagnosis, and the second is the operation, and it seems to me that each step is equally difficult. It is important to make a correct diagnosis, and the diagnosis consists not only of making a diagnosis that the retina is detached and that there are holes in the retina, but also of determining the condition of the vitreous, of the retina, of the choroid and other factors which are to decide the operative procedure. Keeping the patient at perfect rest in bed helps a great deal, because part of the subretinal fluid is absorbed and one has opportunity to find out whether there are tears in the retina or a pathologic process in the choroid. To accomplish this same purpose, occasionally one must make a diagnostic scleral puncture.

With regard to operative technic, one must learn to judge how deeply the electrode should penetrate, how many punctures to make, where the punctures should be placed and how much current to use. A weak current is likely to give better results, and a strong current is apt to defeat the purpose of the operation.

Adequate drainage is important. There is not much danger of removing normal vitreous through a small opening, because healthy vitreous will not pass through a small opening. One should try to remove all subretinal fluid, which is watery and therefore escapes easily.

DR. JOHN W. BURKE: I should like to ask Dr. Knapp what voltage he considers most suitable.

DR. T. B. HOLLOWAY: Does Dr. Knapp find it is difficult exactly to control the current?

DR. SANFORD R. GIFFORD: I should like to mention that Dr. Clifford B. Walker has devised very ingenious electrodes. The Safar electrodes are little buttons which come off easily, and to which it is difficult to apply the current. The Walker electrodes have spiral openings, and they are fastened together so that any number may be applied, and it is easy to apply the current. He has also diathermy electrodes devised for low current.

DR. ARNOLD KNAPP: Confinement of the patient to bed depends somewhat on the location of the tear. As a rule, the period in bed is two weeks, and that for binocular bandages ten days. If the detachment is large and confined to the upper half of the globe, the head is lowered to secure the aid of gravity.

Drainage of subretinal fluid is an important feature, but there again it depends on the type of detachment with which one is dealing. Flat detachments do not seem to require particular care. Balloon-shaped detachments require drainage, which can be facilitated by introducing at the end of the operation cold electrodes to enlarge the openings in the sclera, or by aspiration of some of the subretinal fluid by means of a syringe.

Dr. Schoenberg emphasized two important points, a careful examination of the eye and exact location of the tear.

To control the current, we usually apply enough for the amount of amperage desired for the number of electrodes used.

THE KAHN REACTION IN THE AQUEOUS HUMOR

ITS RELATION TO SYPHILIS OF THE EYE

F. BRUCE FRALICK, M.D.

ANN ARBOR, MICH.

The relatively recent developments in microchemical laboratory technic have stimulated renewed interest in the characteristics of the intra-ocular and other body fluids. Rapid strides have been made in the studies of the constituents of the aqueous and vitreous humors in health and in disease. With the publication by Wassermann and his associates,[1] in 1906, of his serologic reaction for syphilis a new field of investigation was opened. The blood and then the spinal fluid occupied medical investigators in determining the specificity and adaptability of this test for syphilis.

SEROLOGIC REACTIONS IN OTHER BODY FLUIDS

Investigations of the serologic reactions of other body fluids have been made. Wile [2] found that in a large percentage of cases of syphilis the urine contains substances which behave in the same way as the antibodies in the blood serum of the same patient. In general, the Wassermann reaction is more striking and appears earlier in the serum than in the urine. He found that at times a positive reaction is present in the blood and not in the urine, and vice versa. Bab [3] investigated the milk of syphilitic women and found that here also the complement-binding substances were demonstrable.

From the Department of Ophthalmic Surgery of the University of Michigan Medical School.

This study was made possible through the John E. Weeks Scholarship for Research in Ophthalmology.

Read at a meeting of the American Academy of Ophthalmology and Oto-Laryngology, Sept. 20, 1933.

1. Wassermann, A.; Neisser, A., and Bruck, C.: Eine serodiagnostische Reaction bei Syphilis, Deutsche med. Wchnschr. **32**:745, 1906.

2. Wile, U. J.: Comparative Experiments on the Presence of Complement Binding Substances in the Serum and Urine of Syphilitics, J. A. M. A. **51**:1142 (Oct. 3) 1908.

3. Bab, H.: Ueber bacteriologische und serodiagnostische Paralleluntersuchungen, Deutsche med. Wchnschr. **33**:1233, 1907.

Buschke,[4] Serra-Costa,[5] Franchi,[6] von Wikullil [7] and others investigated the relation between the Wassermann reaction of the blood and that of the fluid from a blister produced by a cantharides plaster. They found that the reactions were practically identical except that that of the blister fluid was less intense.

Reschke,[8] Schlesinger,[9] Todd,[10] Hoffman,[11] Chesney, Kemp and Boetjer,[12] Kling [13] and others studied the serologic reaction of the joint fluid in syphilitic arthritis. They claimed that positive reactions of the joint fluid occur in the presence of a negative Wassermann reaction of the blood. They stated that a positive Wassermann reaction in the joint fluid in the presence of a negative reaction in the blood occurs in patients who have had insufficient antisyphilitic therapy. They concluded, therefore, that it is necessary to test both the blood and the joint fluid to rule out syphilis as the cause of arthritis in doubtful cases.

Similar findings were reported by McCrae and Caven [14] in tertiary syphilis of the liver with ascites. They recommended that the Wassermann reaction be tried on the ascitic fluid in every doubtful case with ascites. The Wassermann test of the blood is important if positive, but as in a considerable number of cases of hepatitis with ascites the patients do not give a positive reaction of the blood, because of previous antisyphilitic therapy, no great weight should be given to a negative report until the Wassermann reaction of the ascitic fluid has been determined.

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4. Buschke, A.: Untersuchung von Kantharidenblasen bezüglich Pirquetscher und Wassermannscher Reaktion, Med. Klin. **18**:593 (May 7) 1922.

5. Serra-Costa, P.: Significato del valore della reazione di Wassermann praticata sul siero da vescicante, Riforma med. **41**:913 (Sept. 28) 1925.

6. Franchi, F.: Rapporto fra la reazione Wassermann locale e la reazione Wassermann sul siero di sangue e sul liquido di bolla, Gior. ital. di dermat. e sif. **71**:1063 (Aug.) 1930.

7. von Wikullil, L.: Ueber die Wassermann Reaktion aus Gewebsflüssigkeit, München. med. Wchnschr. **78**:708 (April 24) 1931.

8. Reschke, K.: Zur Diagnose der Gelenksyphilis, Arch. f. klin. Chir. **3**:597, 1918-1919.

9. Schlesinger, H.: Arthrolues und ihre Therapie, in Syphilis und innere Medizin, Berlin, Julius Springer, 1925, pt. 1.

10. Todd, W. H.: Syphilitic Arthritis, Brit. J. Surg. **14**:260 (Oct.) 1926.

11. Hoffman, V.: Syphilis der Gelenke, in Jadassohn, J.: Handbuch der Haut-und Geschlechtskrankheiten, Berlin, Julius Springer, 1928, vol. 17, pt. 3, p. 213.

12. Chesney, A.; Kemp, J., and Boetjer, F.: An Experimental Study of the Synovial Fluid of Patients with Arthritis and Syphilis, J. Clin. Investigation **3**:131, 1926.

13. Kling, D. H.: Wassermann Reaction in Joint Fluids, Am. J. Syph. **13**:596 (Oct.) 1929. Kling, D. H., and Pincus, J.: Further Studies on Wassermann Reaction in Joint Fluid, ibid. **15**:376 (July) 1931.

14. McCrae, T., and Caven, W. R.: Tertiary Lues of the Liver, Am. J. M. Sc. **172**:781 (Dec.) 1926.

Considerable work has been done by Bessemans, Van Canneyt and Vincke [15] on the aqueous humor of normal and syphilitic rabbits. Spirochetes were injected into the testes and into the traumatized cornea, thereby producing primary corneal lesions, generalized syphilis or both. These authors were not able to elicit a positive reaction in the aqueous humor in any of their series, even in the presence of a positive reaction of the blood. However, by activating the aqueous humor by the injection of distilled water into the anterior chamber, they were able to obtain positive reactions on repuncturing the cornea.

A review of the literature since Wassermann's contribution revealed only one reference to the serologic reaction of the aqueous humor in man. While discussing puncture of the anterior chamber as a diagnostic and therapeutic measure, von Lussich-Malkovich [16] mentioned having obtained positive Wassermann reactions in the aqueous humor in three patients, one of whom had a negative Wassermann reaction of the blood. No mention was made of the associated ocular condition.

The foregoing résumé of the interest in the serologic reactions of the various body fluids demonstrates how far behind ophthalmologists are in investigating the reactions of the aqueous humor in syphilitic patients.

The Kahn [17] test has been employed at the University of Michigan Hospital since 1928 as the standard method for the serodiagnosis of syphilis. The superiority of this reaction over the Wassermann test has recently been emphasized in reports of the League of Nations Health Organization. This committee arranged two competitive international conferences on tests for syphilis in which authors of internationally renowned methods or modifications of methods participated. One of these conferences was held at Copenhagen in 1928, and the other at Montevideo in 1930. The official report of the Montevideo

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15. Bessemans, A.; Van Canneyt, J., and Vincke, I.: L'humeur aqueuse dans las syphilis expérimentale du lapin, Compt. rend. Soc. de biol. **104**:341 (May 23) 1930. Van Canneyt, J.: Réactions de Wassermann sur l'humeur aqueuse du lapin normal ou syphilitique avant et après des injections dans la chambre antérieure, Bull. Soc. belge d'opht., 1931, no. 63, p. 103. Bessemans, A., and Van Canneyt, J.: La réaction de Wassermann et l'humeur aqueuse du lapin normal ou syphilitique. Activation par injection d'eau distillée dans la chambre antérieure, Compt. rend. Soc. de biol. **108**:1181 (Jan. 8) 1932.

16. von Lussich-Malkovich: Nadelpunktion der vorderen Augenkammer als diagnostisches und therapeutisches Hilfsmittel, Ber. ü. d. Versamml. d. deutsche ophth. Gesellsch. **47**:288, 1928.

17. Kahn, R. L.: The Kahn Test, A Practical Guide, Baltimore, Williams & Wilkins Company, 1928.

conference contains the following statement:[18] "The majority of serologists taking part in the Montevideo conference agreed that, in the hands of Professor Kahn himself, the Kahn standard test, which (as was the case also at the Copenhagen conference) proved to be absolutely specific and extremely sensitive, was the best of those demonstrated at the conference."

The marked sensitivity of the Kahn reaction makes this test especially desirable in investigations of the serologic reactions of the aqueous humor when only small quantities of fluid are available. A limited number of clinical cases were therefore studied in an attempt to obtain information as to the Kahn reaction of the aqueous humor in relation to the various disease processes and to correlate these reactions with those of the blood and spinal fluid.

EXPERIMENTAL PROCEDURE

The aqueous humor was withdrawn by puncturing the cornea at the avascular limbus with a no. 27 needle, care being taken not to traumatize the iris or lens. By this means variable amounts of aqueous were obtained, ranging from 0.12 to 0.38 cc. The reactions of the blood and spinal fluid were obtained at the same time for comparison. When possible, the samples were obtained during an acute attack of the ocular condition and before antisyphilitic therapy had been instituted. All the patients were subjected to a careful slit-lamp examination to determine the presence or absence of an anterior uveal disease.

Briefly, the technic of the Kahn tests is as follows:

1. Standard Kahn test with serum. This is the regular three tube test, in which three different proportions of serum to antigen are employed. The blood is heated for thirty minutes at 56 C. A standard antigenic suspension is measured into each of three small tubes in amounts of 0.05, 0.025 and 0.0125 cc., respectively, and 0.15 cc. of serum is added to each tube. The tubes containing the antigen-serum mixtures are shaken for three minutes, and in order to facilitate the reading of results, 1 cc. of physiologic solution of sodium chloride is added to the tube containing the largest amount of antigenic suspension and 0.5 cc. to the two remaining tubes. The formation of a precipitate denotes a positive reaction, which is read on the basis of $++++$, $+++$, $++$ and $+$ and $\pm$ respectively. When the reaction is negative the fluid appears opalescent and clear.

2. Standard Kahn test with spinal fluid. This test involves the preparation of a concentrated solution of spinal fluid globulins. Into a small tube, 1.5 cc. of clear spinal fluid is measured, and to this is added an equal amount of a saturated solution of ammonium sulphate to precipitate the globulins. The fluids are thoroughly mixed, heated for fifteen minutes at 56 C., and centrifugated at high speed for fifteen minutes. The precipitated spinal fluid globulins are then packed in the form of a sediment at the bottom of the tube. The supernatant fluid is discarded. To the globulin sediment is then added 0.15 cc. of physiologic solution of sodium chloride; the globulin is readily dissolved. Into another small tube 0.01 cc. of a standard antigenic suspension is measured, and to this the 0.15 cc. of concentrated globulin solution is transferred. The tube is then shaken at the

18. Report of the Laboratory Conference on Sero-Diagnosis of Syphilis, Montevideo, Geneva, League of Nations, Health Organization, 1931, p. 17.

standard speed for four minutes, after which 0.5 cc. of physiologic solution of sodium chloride is added to the completed test. The results are read on the same basis as in the test with serum.

3. Kahn test with aqueous humor. This test is limited by the amount of aqueous humor available. The total volume of aqueous humor is measured by means of a graduated capillary pipet or, better, by means of a 0.5 cc. Luer syringe used in withdrawing the aqueous at the time of the puncture of the anterior chamber. To this fluid is added an equal volume of a saturated solution of ammonium sulphate, and the globulins are precipitated in the same manner as in the test of the spinal fluid. To the globulin sediment is then added physiologic solution of sodium chloride equivalent to one tenth the original amount of aqueous humor. The tube is gently shaken to dissolve the globulins, and into the same tube is measured an amount of standard antigenic suspension equal to one tenth the volume of globulin solution, so that the final proportion of globulin solution to antigenic suspension is 10:1. The mixture is shaken for four minutes at the standard speed, and a drop of physiologic solution of sodium chloride is added before the result is read. The results are read on the basis of the presence or absence of a precipitate.

THE KAHN REACTION IN THE AQUEOUS HUMOR IN HEREDITARY SYPHILIS

The accumulated knowledge of the production of positive Kahn reactions in the spinal fluid may allow me to put forth the hypothesis that a positive reaction in the aqueous humor may be expected when the tissues from which that fluid is derived are syphilitic. Thus one would expect to obtain a positive reaction in the aqueous humor in syphilitic iridocyclitis and a negative reaction in nonsyphilitic involvement of these tissues. It is more difficult to predict the reaction in the aqueous humor when the syphilitic process is in the optic nerve, the choroid or the retina because of the uncertainty as to whether the syphilitic antibodies reach the aqueous chamber. When the inflammatory process is of such a character that the vitreous becomes degenerated, one may logically expect the antibodies to reach the aqueous chamber more easily and thereby give a positive reaction. The reactions in the first nine cases of hereditary syphilis appear to be compatible with the first part of my premise, while the last three cases seem to fall in the group considered in the second part.

It is interesting to note that in all of the patients in this group save one the aqueous humor gave Kahn reactions corresponding to the findings in the blood. The patient in case 9 was first seen with active interstitial keratitis and a positive Wassermann reaction of the blood and spinal fluid ten years before my recent studies were undertaken. He has been given antisyphilitic therapy since that time. It would have been interesting to determine the Kahn or Wassermann reaction of the aqueous humor when he was first seen and to ascertain at what point the reaction in the aqueous humor became negative in relation to the reactions of the blood and spinal fluid. From the evidence presented

by the other patients with hereditary syphilis, it might be supposed that the aqueous in this patient also was positive before antisyphilitic therapy was instituted, but that as a result of adequate therapy it now gives a negative reaction, as do the blood and the spinal fluid. As I

TABLE 1.—*The Kahn Reaction in the Aqueous Humor in Patients with Hereditary Syphilis*

Case	Age	Kahn Reaction: Aqueous	Kahn Reaction: Blood	Kahn Reaction: Spinal Fluid	Previous Antisyphilitic Therapy	Clinical Diagnosis
1	20	Pos.	4+	Neg.	None	Acute iridocyclitis, left eye; syphilitic hydrarthrosis
2	6	Pos.	4+	1+	None	Acute interstitial keratitis; acute iridocyclitis, left eye
3	19	Pos.	4+	4+	1 intravenous injection of arsphenamine (0.2 Gm.); 1 intramuscular injection of bismuth salicylate (0.13 Gm.)	Acute interstitial keratitis; acute iridocyclitis, left eye; neurosyphilis
4	15	Pos.	4+	Neg.	None	Acute interstitial keratitis; acute iridocyclitis, left eye
5	21	Pos.	4+	Neg.	3 intravenous injections of arsphenamine (0.4 Gm.); 2 intramuscular injections of bismuth salicylate (0.13 Gm.); potassium iodide and mercury rubs for 1 month	Acute interstitial keratitis; acute iridocyclitis, left eye
6	15	Pos.	4+	±	None	Acute interstitial keratitis; acute iridocyclitis, left eye
7	21	Pos.	4+	Neg.	None	Acute interstitial keratitis; acute iridocyclitis, right eye
8	5	Pos.	4+	4+	9 intravenous injections of arsphenamine (0.05 Gm.); mercury rubs for 12 months	Acute interstitial keratitis; acute iridocyclitis, left eye
9	20	Neg.	Neg.	Neg.	12 intravenous injections of arsphenamine (0.3 Gm.); 1 intramuscular injection of bismuth salicylate (0.13 Gm.); potassium iodide and mercury rubs for 18 months	Old interstitial keratitis, left eye
10	17	Pos. Pos.	4+	4+	1 intravenous injection of arsphenamine (0.3 Gm.); 1 intradural injection of arsphenamine (0.0003 Gm.); 1 intramuscular injection of bismuth salicylate (0.13 Gm.)	Primary optic atrophy, left eye; juvenile tabes
11	18	Pos.	4+	4+	6 intravenous injections of arsphenamine (0.3 Gm.); 4 intradural injections of arsphenamine (0.0003 Gm.); 5 intramuscular injections of bismuth salicylate (0.13 Gm.)	Secondary optic atrophy; pigmentary chorioretinitis, left eye; juvenile dementia paralytica
12	19	Neg.	4+	Neg.	None	Secondary optic atrophy; old chorioretinitis, left eye

realized that my laboratory methods are not infallible, especially when only small amounts of aqueous are obtainable, the puncture of the anterior chamber was repeated in case 10 ten weeks after the first puncture, and a positive reaction was again elicited. It has not yet been possible to repeat the punctures in cases 11 and 12.

THE KAHN REACTION IN THE AQUEOUS HUMOR IN ACQUIRED SYPHILIS

In table 2 are listed the cases of acquired syphilis investigated. The amounts of drugs used in the therapy are omitted in some instances,

TABLE 2.—*The Kahn Reaction in the Aqueous Humor in Patients with Acquired Syphilis*

Case	Age	Kahn Reaction			Previous Anti-syphilitic Therapy	Clinical Diagnosis
		Aqueous	Blood	Spinal Fluid		
13	33	Pos.	4+	Neg.	None	Acute iridocyclitis, left eye; secondary syphilis
14	17	Pos.	4+	Neg.	None	Acute papillary iritis, left eye; secondary syphilis
15	48	Pos.	3+	Neg.	None	Acute iridocyclitis, right eye; latent syphilis
16	37	Pos. Pos.	Neg.	Neg.	6 intravenous injections of arsphenamine; 26 intramuscular injections of bismuth salicylate	Acute recurrent iridocyclitis, right eye
17	24	Pos.	4+	Neg.	Intravenous injections of arsphenamine; intramuscular injections of bismuth salicylate	Acute iridocyclitis, left eye; secondary syphilis
18	25	Pos.	4+	?	None	Acute iridocyclitis, left eye; latent syphilis
19	54	Neg.	±	Neg.	3 intramuscular injections of bismuth salicylate (0.13 Gm.)	Senile cataract, left eye; latent syphilis
20	63	Neg.	2+	Neg.	3 intravenous injections of arsphenamine (0.5 Gm.)	Glaucoma simplex, right eye; latent syphilis
21	58	Neg.	4+	4+	14 intramuscular injections of bismuth salicylate (0.13 Gm.)	Chronic inflammatory glaucoma, left eye; tabes dorsalis
22	31	Neg.	4+	4+	Intravenous injections of arsphenamine	No pathologic condition in left eye; neurosyphilis
23	28	Neg.	4+	4+	9 intravenous injections of arsphenamine (0.3 Gm.); 6 intradural injections of arsphenamine (0.0003 Gm.); 14 intramuscular injections of bismuth salicylate (0.13 Gm.)	No pathologic condition in right eye; tabes dorsalis
24	57	Pos. Neg.	4+	4+	None	Retinal arteriosclerosis in right eye; neurosyphilis
25	42	Neg.	4+	Neg.	None	Papilledema in left eye; gumma of brain
26	53	Neg.	4+	?	1 intramuscular injection of bismuth salicylate (0.13 Gm.)	Primary optic atrophy, right eye; neurosyphilis
27	38	Neg.	4+	4+	2 intravenous injections of arsphenamine; 15 intramuscular injections of bismuth salicylate	Primary optic atrophy, right eye; tabes dorsalis
28	27	Neg.	4+	4+	None	Primary optic atrophy, right eye; dementia paralytica

since a few of the patients were treated elsewhere before being seen by me.

Case 16 deserves special consideration owing to the fact that the Kahn reactions of the blood and the spinal fluid were negative. The patient was admitted to the hospital because of gonorrheal arthritis and

chronic prostatitis. He denied that he had ever had syphilis or received treatment for syphilis. While he was in the hospital, acute monocular iridocyclitis developed. He had had several previous attacks in this eye, starting ten years before, but none during the past five years. Because of the prostatitis and arthritis, the negative Kahn reactions of the blood and spinal fluid and the absence of focal infection elsewhere, the iritis was classified as being probably due to gonorrhea. However, when the Kahn reaction in the aqueous humor from this eye was found to be positive, the patient admitted that he had had a positive Wassermann reaction of the blood and a secondary rash eleven years previously and had received intravenous and intramuscular injections with a resultant negative Wassermann reaction. I feel that this case is one

TABLE 3.—*The Kahn Reaction in the Aqueous Humor in Nonsyphilitic Controls*

Case	Age	Kahn Reaction			Previous Antisyphilitic Therapy	Clinical Diagnosis
		Aqueous	Blood	Spinal Fluid		
29	49	Neg.	Neg.	Neg.	None	Acute iridocyclitis, left eye
30	34	Neg.	Neg.	Neg.	None	Acute iridocyclitis, right eye
31	34	Neg.	Neg.	Neg.	None	Acute iridocyclitis, right eye
32	31	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, left eye
33	31	Pos.	Neg.	Neg.	None	Acute recurrent iridocyclitis, right eye; uveal cataract, right eye
34	53	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, right eye
35	57	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, left eye
36	14	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, left eye
37	19	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, left eye
38	42	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, right eye
39	43	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, right eye
40	59	Neg.	Neg.	Neg.	None	Acute recurrent iridocyclitis, left eye
41	22	Neg.	Neg.	Neg.	None	Acute uveitis, left eye
42	57	Neg.	Neg.	Neg.	None	Chronic uveitis, left eye
43	39	Neg.	Neg.	Neg.	None	Quiescent iridocyclitis, left eye
44	45	Neg.	Neg.	Neg.	None	Chronic uveitis, left eye
45	14	Neg.	Neg.	Neg.	None	Old eczematous keratoconjunctivitis, left eye
46	27	Neg.	Neg.	Neg.	None	Primary optic atrophy; interstitial keratitis, right eye
47	17	Neg.	Neg.	Neg.	None	Primary optic atrophy, left eye

of recurrent syphilitic iridocyclitis in a patient who has received insufficient antisyphilitic therapy. To check the Kahn reaction in this case, another puncture of the anterior chamber was made twelve weeks later, and the reaction of the aqueous humor was still positive. In case 24, the first puncture of the anterior chamber gave a positive reaction, but when the puncture was repeated one month later, the reaction was negative. The Kahn reactions in the aqueous humor in the patients with acquired syphilis further strengthens the hypothesis already stated.

THE KAHN REACTION OF THE AQUEOUS HUMOR IN NONSYPHILITIC CONTROLS

Keeping case 16 (table 2) in mind, one may then consider the cases listed in table 3, in which no history of syphilis or clinical evidence of syphilis could be elicited. In this group of nineteen cases, the blood

and spinal fluid consistently gave negative Kahn reactions. There was only one exception (case 33) to the finding of a negative reaction of the aqueous humor to conform to the negative reactions of the blood and spinal fluid. This patient admitted that he had had repeated gonorrheal infections but said that he had no knowledge of a syphilitic infection and had received no antisyphilitic therapy. Other etiologic studies gave negative results aside from the finding of an abscessed tooth. Seven weeks later the puncture of the anterior chamber was repeated, and the Kahn reaction was again positive. A course of antisyphilitic therapy is now being given.

CONCLUSIONS

The hypothesis is stated that a positive Kahn reaction in the aqueous humor may be expected when the tissues from which that fluid is derived are syphilitic and that a negative reaction may be expected with a nonsyphilitic involvement of these tissues.

Forty-seven patients were subjected to puncture of the anterior chamber, and the Kahn reactions in the aqueous humor, the blood and the spinal fluid were determined.

The findings seem to substantiate the hypothesis stated.

The most interesting exception to these findings can be expected in inadequately treated syphilitic patients showing iridocyclitis, in whom the reaction of the aqueous may be positive and that of the blood and of the spinal fluid negative.

From the cases investigated which showed involvement of the posterior part of the uvea or of the optic nerve, no means of prediction as to the Kahn reaction in the aqueous humor could be evolved.

No correlation was observed between the Kahn reactions of the spinal fluid and those of the aqueous humor.

The Kahn test of the aqueous humor might be used as a differential diagnostic procedure in certain cases of uveitis of obscure etiology.

RETINAL DETACHMENT IN TOXEMIAS OF PREGNANCY

REPORT OF A CASE

MORRIS JAFFE, M.D.
NEW YORK

It is well known that changes occur in the retina in the toxemias of pregnancy. The picture of the fundus oculi depends to a great extent on the type of toxemia. From a review of the literature it is apparent that in primary toxemias (eclampsia and preeclampsia) in which there is no underlying nephritic condition the predominant picture is that of edema of the retina. Detachment of the retina is not altogether rare in eclampsia, and it would probably be found to occur even more often if routine ophthalmoscopic examinations were made. In addition to the retina, the tendency to edema formation manifests itself in the brain, the kidneys and the liver. Pillat [1] attributed the edema to the circulating toxins, which have a special affinity for these organs. Stander and Peckham [2] found that in primary eclampsia the main picture is edema of the retina which is localized about the optic nerve, but which may later advance to a serous detachment. In cases of chronic nephritis complicating pregnancy, they found the picture of retinitis nephritica, with advanced arteriosclerotic changes, hemorrhages, papilledema, massive retinal exudates and the star-shaped figure surrounding the macula. Retinal detachment, flat or globular, may also occur in these cases.

Differential diagnosis of the various types of pregnancy toxemia is often difficult. The symptoms, physical findings and laboratory data are common to all. However, the ophthalmoscope often furnishes valuable information regarding the presence of antecedent hypertension or renal disease. In addition, evidences of recent, purely toxic changes can, at times, be discovered in the fundus.

Corwin and Herrick [3] reported two cases of serous detachment of the retina without any statement as to their termination. Most of the reports in the literature are of single cases. The first case was reported and described by von Graefe [4] in 1855. In 1902 Helbron [5] collected the reports of twenty cases, including one under his own observation.

1. Pillat, A.: Chinese M. J. **46**:149, 1932.
2. Stander, H. J., and Peckham, C. H.: Am. J. Obst. & Gynec. **14**:313, 1927.
3. Corwin, Jean, and Herrick, W. W.: Am. J. Obst. & Gynec. **14**:783, 1927.
4. von Graefe, Albrecht: Arch. f. Ophth. **2**:202, 1855.
5. Helbron, J.: Berl. klin. Wchnschr. **39**:68 and 103, 1902.

Moore [6] added five more cases of detachment of the retina. In 1921 Schiötz [7] collected the reports of fifty cases, including six cases of Clapp [8] and seven of his own. Blake [9] reported another case in 1922. Since then Benedict and Mussey,[10] Gardner,[11] Hill [12] and Schaanning [13] have reported individual cases. In Hill's [12] patient, the detachment of the retina recurred in a subsequent pregnancy. Following her first pregnancy reattachment of the retina was spontaneous and complete. After the second confinement spontaneous reattachment occurred only in one eye. Fry [14] reported two cases in 1929. Both cases occurred in eclamptic patients. Reattachment of the retina took place spontaneously. He analyzed twenty-one cases from the literature, including his own two cases, and found that retinal detachment is usually bilateral. In fifteen cases the condition was bilateral and in six unilateral. It occurs with equal frequency in primiparas and multiparas. Spontaneous reattachment took place in eighteen of twenty-one cases reported. Some degree of papillitis and neuroretinitis was present in most of the cases. The prognosis in regard to vision depends on the extent of the residual changes in the retina. Richardson [15] reported another case in the same year. The involvement was bilateral. Twenty-four hours following delivery, almost complete loss of vision developed owing to complete detachment. Richardson resorted to trephine of the sclera and chorio-dialysis, with excellent results.

I wish to report a case of detachment of the retina in preeclampsia, which I had the privilege to observe in the obstetric service of the Morrisania City Hospital.

REPORT OF CASE

A white woman, aged 26, was admitted to the Morrisania City Hospital on Feb. 7, 1933. She was in the seventh month of her first pregnancy. The family history and the past history were irrelevant.

The present illness began two weeks before admission, when the patient noticed a gradual swelling of her feet. She regularly attended the prenatal clinic, but no abnormalities of her blood pressure or urine were found. Two days before admission she was seized suddenly with severe headache and epigastric pains.

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6. Moore, R. F.: Roy. London Ophth. Hosp. Rep. **20**:262, 1915-1916.
7. Schiötz, I.: Klin. Monatsbl. f. Augenh. **67**:66, 1921.
8. Clapp, C. A.: Am. J. Ophth. **2**:473, 1919.
9. Blake, E. M.: Bilateral Detachment of Retina in Nephritis of Pregnancy, Arch. Ophth. **51**:586 (Nov.) 1922.
10. Benedict, W. L., and Mussey, R. D.: Am. J. Ophth. **6**:268, 1923.
11. Gardner, M.: M. J. Australia **1**:477, 1923.
12. Hill, E. H.: Bilateral Detachment of Retina in Two Successive Pregnancies, Arch. Ophth. **53**:137 (March) 1924.
13. Schaanning, G.: Norsk mag. f. lægevidensk. **86**:136, 1925.
14. Fry, W. E.: Extensive Bilateral Detachment in Eclampsia, with Complete Reattachment: Report of Two Cases, Arch. Ophth. **1**:609 (May) 1929.
15. Richardson, S.: J. Florida M. A. **16**:266, 1929.

On admission to the hospital, the patient was fully conscious. There was moderate edema of the legs and the face. The blood pressure was 170 systolic and 110 diastolic. Urinalysis showed: albumin, 4 plus; a moderate number of hyaline and granular casts, and a few erythrocytes. Examination of the fundi at this time revealed hyperemic and blurred disks and a small hemorrhage in the left eye. The arteries and veins were tortuous, but there was no disproportion or signs of arteriosclerosis.

On February 10, labor was induced prematurely by a Vorhees bag.

On February 13, further examination revealed a marked increase in the papilledema (the outline of the disks could not be made out). In the nasal portion of the left eye, there was a large bullous detachment of the retina. There was also a smaller flat detachment in the temporal portion of the right eye. Vision was very blurred. The same night, the patient was delivered of a living premature infant.

The day following delivery the blood pressure was 110 systolic and 70 diastolic, and the general condition of the patient was considerably improved. The retinal detachment also improved fairly rapidly. There was no evidence of detachment in the left eye on February 15, and in the right eye on February 17. The optic disks, however, remained swollen. On February 27, a spinal tap was performed and revealed a spinal fluid under pressure of 14 mm. of mercury. A teleoroentgenogram, taken on February 27, showed no cardiac enlargement.

The patient was discharged from the hospital on March 14 (nineteen days post partum). Her blood pressure at that time was 86 systolic and 60 diastolic, and the urine contained a trace of albumin and a few finely granular casts. Edema of the optic disk was still present in both eyes, but was not as marked as before delivery. There were no retinal hemorrhages or exudates.

The patient was observed postnatally in the clinic for patients with toxemia at two week intervals. The blood pressure and urine were normal. The edges of the disks, however, were still blurred on the last examination (May 31). The diagnosis was preeclampsia with detachment of the retina.

SUMMARY

Generalized edema is a common feature in the toxemia of pregnancy. Detachment of the retina is probably one of the manifestations. The brain, the liver and the kidneys are the viscera which frequently become involved. The prognosis in regard to vision is dependent on the residual changes left from the accompanying retinitis and papillitis.

MYDRIATIC GLAUCOMA

A STATISTICAL STUDY

SAMUEL V. ABRAHAM, M.D.
LOS ANGELES

In reviewing the literature on acute glaucoma, particularly in reference to cases following the use of mydriatics, one cannot but be impressed by the few references to mydriatic glaucoma. The term "mydriatic glaucoma," which I believe to be well chosen, was first used by A. Heyl,[1] in 1882, and again by H. Gifford[2] in 1916. I have been unable to find that any other author used either this term or the similar one of "homatropine glaucoma" or "atropine glaucoma." The rarity of case reports suggests that such cases are to be expected as a natural result of the use of mydriatics and therefore remain unreported or that the fear of this form of glaucoma is greater than the rarity of its occurrence justifies.

The term "mydriatic glaucoma" is used here to designate acute glaucoma produced after the use of mydriatics in patients previously free from clinical signs of glaucoma. In determining the possible danger of mydriatics, this limitation would make the presentation more accurate, as the patients with glaucoma who have not been operated on cannot be considered proper subjects for mydriasis, and acute glaucoma occurring in these patients cannot justifiably be called mydriatic glaucoma. It would be helpful to know how many such cases have occurred since attention was first called to the potentialities of mydriatics by Wharton Jones, who was mentioned by H. Derby[3] in 1867. It would be important to know what relation this number bore to the total number of refractions or other examinations under mydriasis.

I have been able to find on record only 26 cases[3a] of acute glaucoma following the use of mydriatics, this number including case reports and cases presented in discussions on this subject. This does not include any known case of glaucoma in which an acute attack was precipitated

1. Heyl, A. G.: Acute Glaucoma, Am. J. M. Sc. **83**:398, 1882.

2. Gifford, H.: Miotics as Adjuvants to Mydriatics, J. A. M. A. **67**:112 (July 8) 1916.

3. Derby, H.: Mydriatics in Glaucoma, Tr. Am. Ophth. Soc. **1**:35, 1867.

3a. This number does not include the cases reported by M. Standish (Ophth. Rec. **11**:243, 1902). Most of his cases occurred in glaucomatous patients. Because of the possibility that some of these cases at least were reported by other men, it was thought best not to include his reports. Actually cases which perhaps should be mentioned as probable instances of mydriatic glaucoma were as follows: 4 due to atropine, 2 to homatropine, 1 to cocaine, 1 to scopolamine and 1 to duboisine.

by the use of a mydriatic. Among the 26 cases there were 2 in which there was a family history of glaucoma (Weeks,[4] Hodges[5]) and 3 others in which the patient's history was suggestive of glaucoma (Woods,[4] Ring,[6] Friedman[7]). In many of the remaining 21 cases much information was missing. There were few cases in which the question of the presence of glaucoma or of a glaucomatous background was definitely eliminated. The number of published cases which could truly come under the classification of mydriatic glaucoma would probably be considerably less than is suggested.

There is no definite indication of the number of cases of mydriatic glaucoma reported and the total number of mydriatic refractions and examinations made by the authors presenting these cases. Gifford[2] believed that an incidence of 1 in 10,000 cases was a conservative estimate. W. W. Kahn[4] had no case in twenty years of practice. J. E. Weeks[4] had no private case of acute glaucoma following the use of homatropine in twenty-five years. W. H. Wilder[4] advised combating the fear of mydriatics, and long before this Streatfield[8] said: ". . . the scare about artificial production of glaucoma . . . is much exaggerated. . . ."

In order to gain a better idea of the relationship between the number of patients examined under mydriatics or cycloplegics and the number of cases of acute glaucoma produced, questionnaires were sent to ophthalmologists of the country. With few exceptions these men were chosen from the list of ophthalmologists certified by the American Board for Ophthalmic Examinations. They were chosen because of their experience, training and reputation. The field was by no means completely covered. Sufficient replies were received, however, to indicate a relative uniformity of opinion on certain points and a marked lack of unity on others.

Questionnaires were sent to two hundred and seven ophthalmologists. Of a total of eighty-two who replied, three gave no data; three used no cycloplegics; six used them in less than 5 per cent of their cases; nine did not use them in patients over 30 or 40 years of age; ten used them in less than 5 per cent of patients over 30 or 40. This left fifty-one reports containing essential data. From the data in the literature it is evident that most cases of mydriatic glaucoma followed the longer-lasting mydriatics and the cycloplegics. It should be kept in

4. In discussion on Gifford, H.: Miotics as Adjuvants to Mydriatics, J. A. M. A. **67**:112 (July 8) 1916.

5. Hodges, F. H.: Arch. Ophth. **14**:42, 1885.

6. Ring, H. W.: A Case of Acute Glaucoma, Tr. Am. Ophth. Soc. **10**:189, 1903.

7. Friedman, A. C. H.: Ophth. Rec. **17**:92, 1908.

8. Streatfield, T. F.: Mydriatics and Glaucoma, Brit. M. J. **2**:193, 1882.

Summary of Cases Reported

Author	Year	No.	Sex	Age, Years	Drug*	Comment
Derby [3]	1867	2	M	43	A	Known glaucoma in both eyes
			F	62	A	Known absolute glaucoma in one eye
Heyl [1]	1882	1	F	55	D	Glaucoma present
Sachs, cited by Gifford: Ophth. Rec. **9**: 329, 1900	1884	1	M	58	H	
Hodges [5]	1885	1	F	44	H	Attack 26 hours after instillation, no operation, brother blind from glaucoma
Rogers: Ophth. Rec. **5**: 421, 1895	1895	1	F	41	H & C	Attack four days afterward, operation
Gifford: Ophth. Rec. **9**: 328, 1900	1900	1	F	21	H	Hyperopia of 4 diopters; no operation
Bennett, cited by Gifford: Ophth. Rec. **9**: 329, 1900	1900	2	F	26	H	Myopia of 6 diopters (juvenile glaucoma); operation
			F	45	H	Attack occurred in office
Shears: Ophth. Rec. **9**: 150, 1900	1900	1	F	52	H	No operation
Ring	1903	1	F	56	E	History suggests glaucoma
Pyle: J. A. M. A. **40**: 1725 (June 20) 1903	1903	1	F	50	H & C	Seen 24 hours later, normal; attack started 36 hours after homatropine and cocaine; operation
Friedman [7]	1908	1	F	37	H	Attack in evening; operation; history of glaucomatous attack 3 or 4 years before under cycloplegia; vision normal without correction
Strickler, in discussion on Friedman [7]	1908	1	F	32	H	No operation
Stevens, in discussion on Friedman [7]	1908	1	..	30	H	No operation
Stevenson: Ophthalmoscope **11**: 73, 1913	1913	1	M	23	H	No operation
Plastinin: Klin. Monatsbl. f. Augenh. **52**: 896, 1914	1914	1	..	..	C	Author also mentions one case following phenacaine in a patient with one eye definitely glaucomatous
Gifford [2]	1916	4	F	30	H	Attack occurred in test period; no operation
			F	65	H	Attack in evening; no operation
			F	44	H	No result from correction; right eye blind; left eye normal
			F	69	H	Left eye, better vision before test; 20/70 (amblyopia ?)
Wilde [4]	1916	1	F	48	H	No operation
Weeks, L. E.[4]	1916	2	F	17	H	Attack in afternoon; operation; mother had glaucoma
					C	No data
Bulson [4]	1916	2	..	..	..	No data
Woods [4]	1916	1	M	49	H	Attack in office; no operation; history of halos obtained after attack
Sutphen: Ann. Ophth. **25**: 730, 1916	1916	1	F	38	A	Attack 9 days after first drops, 7 days after stopping atropine; attack mild until use of more atropine for suspected iritis (synechia?) and then severe attack 6 days later; no operation
Levitt: New York M. J. **106**: 362, 1917	1917	1	F	..	H	No operation

* A = atropine; D = duboisine; H = homatropine; C = cocaine; E = eucatropine.

mind that the incidence of this type of glaucoma as indicated by the reports received would probably be greatly reduced if my question had not unintentionally contained the word "cycloplegic" instead of "mydriatic" in more than half the questionnaires.

By the twenty-nine physicians who used mydriatics and who gave the total number of cases thus examined, there were listed 304,500 cases, or an average of slightly more than 10,500 cycloplegic examinations per man. At this rate, in the group of fifty-one essential reports over one-half million such examinations were made. In this group there were 29 cases of mydriatic glaucoma (with presumably no evidence of glaucoma before examination) making an incidence of 1 in approximately 18,400 cycloplegic examinations. No doubt some of the 29 patients would have shown clinical signs of a glaucomatous nature before mydriasis. In one report 6 or 8 cases were cited, but the data strongly indicated that glaucoma was present in one eye in several of these cases, as the reporter cited the occurrence of acute glaucoma in one eye following the instillation of cocaine during operation on the other eye for glaucoma. In the total of 29 cases, only 4 (in which the premydriatic condition seemed less questionable) were listed for this author; this was the highest number reported (by one other).

Eighteen of these fifty-one authors used a miotic to counteract the effect of the mydriatic in all patients over 30 (some in patients over 40) years of age. These authors reported 8 cases of mydriatic glaucoma, or an incidence of 1 in approximately 24,000 examinations under cycloplegia. This incidence is practically unchanged if one includes the reports of the authors who use a miotic at least 50 per cent of the time. Of the twenty-three ophthalmologists who reported cases of mydriatic glaucoma, none used mydriatics less than 5 per cent of the time, and seven did so in less than 5 per cent of patients over 40 (these seven authors reported 11 cases). Of the fifty-six authors who did not report cases, nine used mydriatics in less than 5 per cent of all refractions, and twenty-two did so in less than 5 per cent of patients over 40.

As the literature shows only 4 cases of mydriatic glaucoma in patients under 30, and as the additional data supplied by those answering the questionnaires indicated no other case in a person under 30 years of age, it is safe to assume that mydriatic glaucoma in patients under 30, even without the use of miotics after mydriasis, is extremely rare. The consideration of the data, then, may be limited to an attempt to determine the incidence of mydriatic glaucoma in patients over 30. The average percentage of cyloplegic refractions in this older group of patients was approximately 25. If one considers that in the average practice about 50 per cent of patients are 30 or over, the number of refractions under mydriasis or cycloplegia in patients above this age would average about 2,600 per man. The incidence of mydriatic glau-

coma in this group would then be 1 case in approximately 4,600 cycloplegic refractions. If one believes that more patients over 30 years of age are seen, the incidence is proportionately decreased, more nearly approaching that given by H. Gifford for all mydriatic examinations.

It seems apparent that mydriatic glaucoma is observed more frequently by ophthalmologists who use mydriatics extensively than by those who use them less, and that the incidence is less if a miotic is used following the mydriatic, as H. Gifford and others have advised. The incidence is less than 0.0217 per cent in the group of patients over 30, and considerably less (near the vanishing point) in patients under 30 years.

In the 26 cases reported in the literature, as previously mentioned, sex or age, or both, were given in 17 cases, in which the patients were reasonably free from suspicion of glaucoma before mydriasis. The group comprising these 17 cases and the 7 added by men answering questionnaires showed 19 females, 3 males and 2 in which sex was not designated. Of these 24, 7 occurred in persons 50 or over (1 in a man); 7 between 40 and 50; 6, between 30 and 40, and 3, between 20 and 30 (1 male). In 1 case the patient's age was not designated. The patient under 20 whose case was reported in the literature was a girl of 17 whose mother had glaucoma. In 20 cases containing definite data on this point, 6 patients with mydriatic glaucoma were operated on in at least one eye, while 14 did not require operation.

CONCLUSIONS

1. Mydriatic glaucoma is more frequent after the use of the longer-lasting mydriatics.

2. Mydriatic glaucoma occurs most frequently in females.

3. The use of a miotic after mydriasis reduces the incidence of this form of glaucoma.

4. The use of a miotic after mydriasis when indicated cannot be considered as an argument against the use of the mydriatic.

5. No miotic is indicated following mydriasis in healthy eyes in patients under 20.

6. The danger of mydriatic glaucoma in children has no basis; and this applies with almost equal force to all persons under 30.

7. If proper precautions are taken in cases with suggestive signs, such as a shallow anterior chamber or a family history of glaucoma, there is no appreciable need to fear the use of mydriatics even in patients over 40 years of age.

8. Among patients over 30, the incidence of mydriatic glaucoma is approximately 0.0217 per cent.

9. The greater tendency toward ciliary hyperemia and ciliary hypertrophy in hyperopes, with possible resultant blockage of outflow and increase of inflow of aqueous in uncorrected or undercorrected cases, and the probability that these are causative factors in explaining the greater incidence of glaucoma in hyperopes are strong arguments in favor of the use of cycloplegics.

USE OF NUPERCAINE IN OPHTHALMOLOGY

MAXWELL K. BOCHNER, M.D.
TORONTO, CANADA

In the last few years a voluminous literature has accumulated on the use of nupercaine, a new anesthetic, in general surgery, obstetrics and gynecology, urology and otolaryngology for surface, spinal and infiltration anesthesia. Only slight reference, however, has been made to its use in ophthalmology, and as far as I am aware no paper in English has appeared concerning its value in ocular therapeutics. This has prompted me to prepare a short summary of my experiences with this anesthetic over a period of three years. The results in the main have been most favorable.

CHEMISTRY AND PHARMACOLOGY

Nupercaine is the hydrochloride of diethylethylenediamide of alpha-butyloxycinchonic acid. It belongs to a group of chemical combinations which until recently were not known to possess anesthetic properties. It does not belong to the same family as cocaine or procaine hydrochloride, but is a derivative of quinoline, which is related to quinine. It forms colorless, odorless and tasteless crystals which conglomerate at 90 C. and liquefy at 97 C. Nupercaine is readily soluble and forms a neutral solution in both alcohol and water. It is best prepared by dissolving it in physiologic solution of sodium chloride, and it should be kept in alkali-free glass containers. Contamination with alkali becomes evident by a cloudiness which readily disappears on the addition of 1 drop of dilute hydrochloric acid to every 100 cc. I have found that the solution is less irritating for instillation in the eye when boric acid is substituted for the dilute hydrochloric acid. The addition of 20 grains (1.2 Gm.) of boric acid to an ounce (30 cc.) of solution prevents contamination with alkali and delays the growth of fungi. The solution can be repeatedly sterilized by boiling without losing any of its activity. For infiltration I prefer to have the solution freshly prepared, adding the epinephrine prior to use.

It has been shown by Uhlmann [1] and others that a 1 : 125,000 solution of nupercaine has a demonstrable anesthetic effect on the rabbit's

Read before the Section of Ophthalmology, Academy of Medicine, Toronto, Canada, Dec. 12, 1932.

1. Uhlmann, F.: Ueber Perkain, ein neues Lokalanaesthetikum, Narkose u. Anaesth. **2**:168, 1929.

cornea, whereas a 1:10,000 solution of cocaine is necessary to produce a similar effect. In order to abolish the corneal reflex within five minutes a minimal concentration of cocaine of 1:1,000 was required, whereas 1:50,000 nupercaine was sufficient. If 1:1,000 solutions are used the anesthetic effect of cocaine lasts fifty-five minutes, whereas nupercaine is effective for one hundred and thirty minutes.

From these laboratory experiments the workers concluded that the anesthetic effect of nupercaine is ten times as strong as that of cocaine, and that its action is much more prolonged. Uhlmann found nupercaine to be four times as toxic as cocaine, but since the dilution used was one-tenth that of cocaine it was relatively much less toxic. More recently, Bond and Bloom [2] found that the resistance of guinea-pigs to subcutaneous injections of nupercaine is more than two and a half times that found by Uhlmann, so that the toxicity is less than twice that of cocaine. They also found, contrary to previous reports, that nupercaine is rapidly detoxicated in vivo and is four times as rapidly eliminated as cocaine. Lipschitz and Laubender [3] showed that the intact skin of a frog (which is in many ways comparable to human mucous membrane) resists the passage of nupercaine into the adjacent tissues, although the sensory nerves of the skin itself are completely anesthetized. It is claimed, therefore, that the risk of poisoning by absorption through the mucous membrane is much less to be feared when nupercaine is used for surface anesthesia than when cocaine is employed. Clinical experience with the use of nupercaine in the eye seems to bear out this claim.

PERSONAL EXPERIENCES

In my early experience with nupercaine, on the recommendation of the manufacturers I used a 2 per cent solution for instillation. Two successive cases of superficial chemical burns of the corneal epithelium led me to dilute the solution to 1 per cent and to substitute boric acid for the dilute hydrochloric acid. During the past fourteen months, for routine office use, I have been employing a 0.5 per cent solution in the following formula:

			Gm. or Cc.
℞	Nupercaine	2 grains	0.12
	Boric acid	20 grains	1.2
	Epinephrine chloride	20 minims	1.2
	physiologic solution of sodium chloride, to make	1 ounce	30

2. Bond, W. R., and Bloom, N.: Studies on the Toxicity of Alpha-Butyloxy-Cinchoninic Acid Diethyl-Etheylene-Diamide Hydrochloride (Nupercaine), J. Lab. & Clin. Med. **16**:447 (Feb.) 1931.

3. Lipschitz, W., and Laubender, W.: Die pharmakologischen Wirkungen des Perkain, eines neuen Lokalanaesthetikuns, Klin. Wchnschr. **8**:1438, 1929.

For surgical use I still employ a 1 per cent solution. The addition of 20 drops of epinephrine per ounce (30 cc.) is essential to counteract the marked vasodilator action. The epinephrine does not seem to influence the anesthetic effect of the nupercaine.

In using the preparation described for instillation in over 2,000 cases I have never had a corneal burn nor have I observed any toxic reaction to the drug. There is a slight smarting sensation after the first drop is instilled, and loss of corneal sensitivity begins within one minute. Five instillations at intervals of three minutes produce complete corneal anesthesia, which lasts from two and a half to three hours, followed by a variable period of relative analgesia. Slight drying of the corneal epithelium occurs, but there is no increase in tension or influence on the pupil.

Cases in Which Nupercaine Was Used

Operation or Treatment	Number of Cases
Cataract extraction	34
Iridectomy for acute glaucoma	6
Corneoscleral trephine	9
Operations on the lacrimal sac	36
Enucleation	14
Operations for pterygium	11
Operations for squint in adults	19
Plastic operations	12
Operations for chalazion	162
Probing and irrigation of the tear duct	344
Removal of foreign bodies	403
Tonometry	742
Treatment of corneal ulcers	54
Painting the lids with silver nitrate or copper sulphate	147
Removing sutures	62
Traumatic injuries to the cornea and conjunctiva	29
Prescription for drops or ointment	119
	2,201

For infiltration, I use a 1:1,500 freshly prepared solution (never stronger than 1:1,000) to which is added 10 drops of epinephrine to every 100 cc. No irritation of the tissues or complications, such as necrosis or delayed healing of the wound, have been observed following injection. To obtain the maximum effect of the anesthetic it is advisable to wait at least ten minutes after the injection before operating.

In my opinion, the great advantage of nupercaine lies in its prolonged action and its low relative toxicity. Both of these factors are due to its slow absorption and rapid detoxication. This has rendered it difficult to obtain deep anesthesia in some surgical cases involving manipulation of the iris unless the instillations were supplemented by a retrobulbar injection. I have operated on both eyes of the same patient at different periods, using nupercaine in one eye and cocaine in the other, and in some cases traction on the iris was felt much more with nupercaine than with cocaine. More recently, I have been able to obtain deep anesthesia with instillation alone by allowing an interval of at least one-half hour between the instillation of the first drop and the operation.

In operations on the ocular muscles instillation anesthesia alone is satisfactory. I find it helpful to supplement the drops by holding a cotton applicator dipped in nupercaine over the exposed muscle for two minutes.

Enucleations can be done almost painlessly ten minutes after the retrobulbar injection of 4 cc. of 1:1,500 nupercaine and the instillation of 5 drops in the conjunctival sac. Moreover, since I have used a tonsil snare for enucleation the field is almost bloodless.

For routine tonometry, the removal of foreign bodies, the removal of sutures, the treatment of corneal ulcers, the treatment of the conjunctiva with silver or copper, the treatment of injuries to the cornea and the use of ethylmorphine hydrochloride in the eye, instillation anesthesia is ideal. For probing or irrigation of the tear ducts, three instillations two minutes apart, followed by the injection of 5 minims (0.3 cc.) into the sac, produce excellent anesthesia in ten minutes, and probing and irrigation can be performed with practically no pain.

The fact that the anesthetic is a nonnarcotic makes it suitable for prescription either in drop or in ointment form for use at home.

A very weak solution is beneficial when added to an eye wash or to the physiologic solution of sodium chloride in the contact glass for conical cornea.

I have found the following ointment helpful in ocular burns, injuries of the cornea and conjunctiva, hay fever, vernal conjunctivitis and herpes and after the removal of foreign bodies and minor operations:

			Gm. or Cc.
℞	Nupercaine	4 grains	0.24
	Boric acid	20 grains	1.2
	Epinephrine chloride	3/200 grain	0.0009
	Anhydrous wool fat	10 drachms	40
	Liquid petrolatum	6 drachms	24
	Oil of bergamot	5 minims	0.3

Many drugs with irritating qualities limiting the strength in which they are used, such as zinc sulphate in angular conjunctivitis and salicylic acid in eczematous conditions of the eyelids, can be advantageously added to the nupercaine ointment in a much greater percentage. This gives the double advantage of greater concentration of the active principle and less irritation. Patients suffering from painful corneal conditions can be kept comfortable by the application of this ointment every three hours.

SUMMARY AND CONCLUSIONS

Nupercaine is a most satisfactory anesthetic for both instillation and infiltration anesthesia in ophthalmologic practice.

The maximum strength should never exceed 1 per cent for instillation and 1:1,000 for infiltration.

The maximum effect does not take place for at least ten minutes after the injection or instillation, and for operations involving traction of the iris an interval of one-half hour should elapse between the instillation of the first drop and the operation unless the instillation is supplemented by a retrobulbar injection.

For instillation at least from 15 to 20 drops of epinephrine hydrochloride per ounce (30 cc.) should be added to overcome the vasodilator effect. The epinephrine does not appear to increase the anesthetic action.

There is only slight drying of the corneal epithelium, and no alteration in tension or influence on the pupil results.

The action is much more prolonged that that of any other anesthetic, and the drug is relatively less toxic. In more than 2,000 cases no toxic reaction was observed when the drug was used in the strength mentioned.

As a non-narcotic it lends itself readily to prescription in combination with other drugs, in the form of either solutions or ointments.

425 Bloor Street, West.

VISUAL RESULTS IN CASES OF INTRA-OCULAR FOREIGN BODY

A STUDY OF TWO HUNDRED AND SEVENTY CASES

WALTER F. DUGGAN, M.D.
NEW YORK

In 1932, Kiehle,[1] after a survey of statistics on foreign bodies, pointed out that the important fact in every case is "how much vision is retained and how long it is retained." He also stated (*a*) that an estimate of vision six months after removal of a foreign body is not particularly significant, (*b*) that perception of light or vision of fingers at a few feet is nothing of which to boast, and (*c*) that the longer the period of time elapsing after removal of the foreign body, the greater is the diminution of vision. After mentioning one author who limited his expressions to "result good," "eye quiet," "excellent recovery," "no subsequent record of vision" and "successful," he stated that interest seems to be lost in these cases in proportion to the square of the time by which they recede into the past. After mentioning the well known fact that no general surgeon considers a cancer cured even when the patient is alive six months after its removal, he concludes by suggesting that as visual function tends to decrease as time elapses after an injury, the final estimate of disability should be delayed at least two years and such cases should be kept under periodic supervision.

With the aforementioned facts in mind, it was decided, at the suggestion of Dr. Arnold Knapp, to investigate a series of cases of intra-ocular foreign body, laying particular emphasis on the visual results. The cases were grouped as: (*a*) cases in which the foreign body was removed within four months of the time of entrance and the patients were followed up less than one year after removal of the foreign body; (*b*) cases in which the foreign body was removed within four months and the patients were followed up for one year or more; (*c*) cases in which the foreign body was in the eye for four months or more before removal, and (*d*) cases in which the foreign body was not removed. (Bulson, quoted by Kiehle, thinks that the results are about the same

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From the Herman Knapp Memorial Eye Hospital.

Read before the Section of Ophthalmology, New York Academy of Medicine, Nov. 20, 1933.

1. Kiehle, Frederick A.: The Aftermath of Cases of Intra-Ocular Foreign Body, Arch. Ophth. **7**:180 (Feb.) 1932.

whether the foreign bodies are removed from the posterior segment or are left in situ, while many ophthalmologists have mentioned the fact that iron salts produce a slow degeneration of intra-ocular structures, especially those of epithelial origin, the macula being particularly vulnerable). Next (*e*) the relation between scleral wounds and detachment of the retina and vitreous exudate was investigated since, in recent papers, Verhoeff [2] and Miles [3] concluded that scleral incisions favored retinal detachment, while Shoemaker [4] and Stieren [5] expressed the belief that the scleral route is preferable for removing a foreign body from the vitreous, Stieren's paper being a direct refutation of Verhoeff's. Finally, the visual results in (*f*) cases of siderosis and in (*g*) cases of an orbital foreign body resulting from a double perforation of the globe were separately considered.

Material was obtained by reviewing the records of all hospitalized cases at the Herman Knapp Memorial Eye Hospital from Jan. 1, 1920, to Dec. 31, 1931. Cases were included in which the foreign body had been removed at some other hospital, the patient subsequently coming to the Herman Knapp Memorial Eye Hospital for further treatment, as well as cases in which the foreign body had been removed prior to 1920, but the patients were readmitted between the dates mentioned for further treatment. Two hundred and sixty-nine cases were obtained from the survey of 10,335 admissions, to which was added 1 case of orbital foreign body (double perforation of the globe) from Dr. Arnold Knapp's practice previous to 1920, making a total of 270 cases. Late results were obtained by investigating the clinical records and by communicating with the various surgeons.

One year was taken as the minimum follow-up period for the definitive series (table 2, 43 cases), since, if two years had been taken, the available cases would have been decreased by almost 50 per cent. Moreover, as it is difficult to find the results even for a follow-up period of one year in the literature, it was felt that even a short series would be of some value for future reference and comparison.

In 1916, Gray Clegg [6] reported the visual results in 130 cases of foreign body in the eye or the orbit. Among these were 20 cases in which the foreign body was removed within a month (a few hours in most of

2. Verhoeff, F. H.: Concerning Magnetic Intraocular Foreign Bodies and Their Removal, Am. J. Ophth. **15**:685, 1932.

3. Miles, H. S.: Foreign Bodies in the Eyeball, Arch. Ophth. **7**:925 (June) 1932.

4. Shoemaker, W. T.: Wounds of the Eyeball, Tr. Am. Ophth. Soc. **17**:115, 1919.

5. Stieren, E.: Magnet Extraction from the Vitreous, Am. J. Ophth. **15**:1120, 1932.

6. Clegg, J. G.: Tr. Ophth. Soc. U. Kingdom **36**:86, 1916.

the cases) and the cases were followed up for one year or longer. Of these 20 patients, 20 per cent had 20/20 vision; 10 per cent, 20/30; 10 per cent, 20/40; 30 per cent, from 20/70 to 20/50; 10 per cent, from 20/200 to 20/100; 15 per cent, less than 20/200, and 5 per cent, no vision. There were 3 cases of traumatic cataract, 7 of aphacia, 2 of secondary membrane, 1 of detachment, 2 of vitreous exudate and 3 of scleral incision. The detachment followed removal of a foreign body through the sclera.

Many groups of statistics were found inadequate for one reason or another. Thus Spratt [7] reported 101 cases in which the visual results were listed as "good" (5/12 or better) and "poor" (less than 5/12). There were at least 13 cases of anophthalmos included in the group having "poor" vision and probably more, because Spratt mentioned that in 27 cases in which steel in the vitreous was removed by the scleral route, there were 8 good results and 19 with poor vision or loss of the eye. The length of time these cases were followed up is not indicated, and minor discrepancies exist between the tabulated results and the accompanying text.

Stieren reported 700 cases followed up from one to twenty-five years. In 15 per cent the eyes were enucleated at once; 5 per cent had vision of 20/20 or better; 22 per cent, from 20/30 to 20/100; 26 per cent, from 20/120 to 20/200, and 32 per cent, from 20/240 to perception of light. The question immediately arises as to whether there were any enucleations not done immediately and also as to the advisability of grouping cases with vision of 20/30 with those with vision of 20/100.

In the present paper, lack of space prevents the inclusion of abstracts of the cases studied as well as many of the tables.

In the cases listed as "unknown," the foreign body was intra-ocular and was not in the anterior chamber, lens, iris or orbit, so it may be considered as having been somewhere in the posterior segment of the eye.

The foreign bodies located in the "orbit" had all caused a double perforation of the globe; in 3 of the cases the foreign body was in the posterior sclera as well as in the orbit. In all of the cases of orbital foreign body, the foreign body was assumed to have been removed from the globe proper at the time of the original injury.

Among the tabulated complications (additional data), "traumatic cataract" includes 2 cases in which an exudate covered the pupillary region of the anterior surface of the lens. Traumatic cataract was listed as a complication even in those instances in which vision was good (20/20 in 1 case) because it was felt that such a lens would prob-

7. Spratt, C. H.: Intraocular Foreign Body: A Review of 101 Cases, Am. J. Ophth. **13**:1079, 1930.

ably react to a relatively slight trauma with a further increase in the lenticular opacity.

"Vitreous exudate" is to be interpreted as meaning a true inflammatory exudate, a hemorrhage or connective tissue which followed either of the former two conditions.

The usual method of removing a foreign body was by way of the anterior chamber. Only when this was impossible or there was a scleral wound was the posterior route used or attempted.

The extreme limits of time during which a foreign body had been in the eye before removal were two hours and twenty years.

The types of foreign bodies found were as follows:

Lead—7 (vitreous, retina, unknown, 1 each; orbit, 4)
Glass—5 (anterior chamber, ciliary body, choroid, 1 each; vitreous, 2)
Nonmagnetic metal—5 (ciliary body, 1; lens, vitreous, 2 each)
Brass—3 (iris, ciliary body, lens, 1 each)
Wood—2 (iris, vitreous, 1 each)
Bronze—1 (vitreous)
Copper—1 (unknown)
Not stated—14 (anterior chamber, 2; vitreous, 8; retina, 1; unknown, 3)
Steel—232 (all remaining cases)

The sexes were represented by 261 males and 9 females. There were 124 right eyes involved and 146 left eyes. In no case were both eyes involved. Two patients had multiple foreign bodies. The first had steel and rust (?) on the iris. The steel was removed ten days after the accident, but the eye continued to be inflamed; so the rust (?) was removed by iridectomy three weeks later. At the time of discharge, vision was 20/40, and there was a faint posterior cortical opacity. Eight years later, vision and opacity were unchanged. The second patient had steel in the posterior chamber and vitreous. The former was removed after five days, but the latter could not be removed even through a scleral incision. A detachment was found five days after the scleral incision. Two years later, the field was markedly contracted and only perception of light was present.

I. CASES FOLLOWED UP LESS THAN ONE YEAR AFTER REMOVAL OF THE INTRA-OCULAR FOREIGN BODY

There were 175 cases which were followed up for from one week to ten and one-half months after removal of the foreign body. However, it was felt that when a foreign body had remained in the eye for four months or more before removal, sufficient degenerative processes might have been set up to make a true picture of the visual results unobtainable.

There were 16 cases in which the foreign body had been in the eye from four months to twenty years before removal; they may be briefly summarized as follows:

Anterior chamber—1 (vision less than 20/200)
Ciliary body—1 (vision less than 20/200)
Lens—5 (vision less than 20/200, 1; 20/200, 1; 20/20, 2; not stated, 1)
Vitreous—3 (vision less than 20/200, 2; 20/20, 1)
Retina—3 (vision less than 20/200, 2; 20/200, 1)
Choroid—1 (vision less than 20/200)
Unknown—2 (vision less than 20/200)

TABLE 1.—*Foreign Body Removed Within Four Months; Cases Followed Less Than One Year After Removal of Foreign Body* *

			Vision								Additional Data							
	Total	Enucleated	None	Less Than 20/200	20/200 to 20/100	20/70 to 20/50	20/40	20/30	20/20	Not Stated	Cataract	Aphacia	Secondary Membrane	Detachment	Vitreous Exudate	Siderosis	Scleral Wound	Phthisis
Anterior chamber..	12	..	..	..	1	..	3	1	5	2	..	..	..	..	..	..	..	..
Iris.....	13	1	..	2	1	..	..	1	7	1	2	..	..	..	..	..	..	..
Posterior chamber.	5	..	..	1	..	..	1	1	1	1	..	1	1	..	..	..	..	..
Ciliary body.....	3	1	..	1	..	..	1	..	..	..	..	..	..	1	..	..	2	..
Lens.....	17	2	..	1	1	3	2	3	1	4	2	12	..	..	..	..	..	..
Vitreous.....	70	20	9	16	1	5	1	5	7	6	8	11	4	4	11	..	12	6
Retina.....	12	2	..	5	..	1	3	..	..	1	2	..	..	..	2	..	4	..
Choroid.....	5	3	..	..	1	1	..	..	..	..	1	..	..	..	2	..	3	..
Unknown.....	12	4	1	..	3	..	1	..	1	2	2	1	..	..	..	..	..	1
Orbit.....	10	5	1	2	..	..	..	..	1	1	1	2	..	3	1	1	2	1
Total.....	159	38	11	28	8	10	12	11	23	18	18	27	5	8	16	1	23	8
Percentage.....		23.9	6.9	17.6	5.0	6.3	7.6	6.9	14.5	11.3								
Percentage in surviving eyes (121).....			9.1	23.1	6.6	8.3	9.9	9.1	19.0	14.9								

* Five detachments were preceded by scleral incision (or wound); 9 vitreous exudates were preceded by scleral incision (or wound); (1 case also showed a detachment).

As is apparent, 62.5 per cent of the 16 patients had vision of less than 20/200, 12.5 per cent had vision of 20/200, 18 per cent had vision of 20/20, and vision was not recorded in 6.3 per cent. Complications included 5 cataracts, 1 secondary membrane, 3 detachments and 3 cases of siderosis.

The remaining 159 cases are tabulated in table 1, with percentages for the total number of cases and for the surviving eyes.

There was 1 case of sympathetic ophthalmia in the foregoing series; the foreign body (location unknown) was removed four days after the injury, and the eye was enucleated for sympathetic irritation in the other eye three and one-half weeks later. In spite of severe iridocyclitis (sympathetic?) in the remaining eye eleven and twelve years later, the final vision was 20/20 two months after the last attack of iridocyclitis.

In the case of siderosis in table 1, the foreign body (steel) measured 11.5 by 1.5 mm. The case was seen seven weeks after the injury, and the eye was enucleated one week later for iridocyclitis. The foreign body was found adherent to the posterior sclera (double perforation), most of the foreign body being in the orbit (case 11, table 6).

II. CASES FOLLOWED UP ONE YEAR OR MORE AFTER REMOVAL OF INTRA-OCULAR FOREIGN BODY

There were 53 cases which were followed up for one year or more after removal of the foreign body. However, among these 53 cases there were 10 in which the foreign body had been in the eye for more than four months before removal (from four and one-half months to one year in 6 cases, and from one and one-fourth to eight years in 4 cases). These 10 cases may be briefly summarized as follows:

Ciliary body—3 (vision less than 20/200, 2; 20/70, 1)
Lens—2 (vision less than 20/200, 1; not stated, 1)
Vitreous—2 (vision less than 20/200, 2)
Choroid—2 (vision less than 20/200, 2)
Unknown—1 (vision less than 20/200, 1)

As is evident, 80 per cent of the 10 patients had vision of less than 20/200, 10 per cent had vision of 20/70, and vision was not stated in 10 per cent. Complications included 2 cataracts, 4 detachments, 1 vitreous exudate and 6 cases of siderosis.

The remaining 43 cases are tabulated in table 2, which is to be considered as the definitive series. Of these 43 cases, the foreign body was removed within six hours in 8 cases and after from one day to one week in 20 cases, from eight days to two weeks in 6 cases, from fifteen days to one month in 3 cases and from five weeks to three months in 6 cases.

The enucleation in this group occurred in a boy of 10 years who received a charge of shot in the face, one of which penetrated the left globe and lodged in the orbit. When first seen one year after the injury, the left eye was blind and phthisical. Roentgen examination revealed a foreign body in the left orbit. In the right eye (in which vision had been failing for six months) vision was 20/200; there were a shallow anterior chamber, an atrophic iris, an irregular pupil, posterior synechiae and a pupillary exudate. There was a faint fundus reflex. Tension varied between 26 and 41 mm. (Schiötz). A diagnosis of sympathetic ophthalmia was made. The left eye was enucleated, and as the patient gave a positive reaction to an intradermal injection of uveal pigment (Woods), he was given injections of pigment for three months. After one month the tension dropped to 23 mm., the fundus

reflex improved, and vision improved to 20/50. This improvement was maintained for the remainder of the three months, at which time the patient returned to Vermont. Information was received three and one-fourth years later that vision was reduced to perception of light, and that the patient had been placed in an institution (case 5, table 6).

In the case of siderosis in table 2, the foreign body (in the vitreous) was removed after two months. Vision was 20/70 before removal and 20/20 one and one-fourth years after removal.

The correlation of the cases in table 2 with Gray Clegg's series is amazing, since 20 (15.4 per cent) of his 130 cases were followed up

TABLE 2.—*Foreign Body Removed Within Four Months; Cases Followed One Year or More After Removal of Foreign Body* *

			Vision							Additional Data							
	Total	Enucleated	None	Less Than 20/200	20/200 to 20/100	20/70 to 20/50	20/40	20/30	20/20	Cataract	Aphacia	Secondary Membrane	Detachment	Vitreous Exudate	Siderosis	Scleral Wound	Phthisis
Anterior chamber	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..
Iris	7	..	..	..	1	..	1	2	3	1	1	..	1	..	..	..	..
Ciliary body	4	..	..	2	..	..	..	1	1	1	1	..	..	..	..	1	..
Lens	4	..	..	1	1	1	1	..	..	1	3	2	..	..	..	..	..
Vitreous	14	..	..	5	2	..	1	2	4	2	7	2	1	2	1	2	..
Retina	4	..	..	..	..	..	..	2	2	..	1	..	1	..	..	1	..
Choroid	3	..	..	..	..	2	..	..	1	1	..	..	..	1	..	1	..
Unknown	2	..	1	1	..	..	..	..	..	..	1	1	..	..	..	..	1
Orbit	4	1	..	3	..	..	..	..	..	..	1	..	..	3	..	..	..
Total	43	1	1	12	4	3	3	7	12	6	15	5	3	6	1	5	1
Percentage		2.3	2.3	27.9	9.3	7.0	7.0	16.3	27.9								
Percentage in surviving eyes (42)			2.4	28.6	9.5	7.1	7.1	16.7	28.6								

* Two detachments were preceded by scleral incision (or wound).

for one year or more, while 43 (15.9 per cent) of the 270 cases discussed in this paper were followed up for one year or more. Apparently patients do not return for follow-up examination any better in England than in the United States.

III. CASES IN WHICH THE FOREIGN BODY WAS IN THE EYE FOR FOUR MONTHS OR MORE BEFORE REMOVAL

There were 16 cases (described in section I) which were followed up less than one year after removal of the foreign body and 10 cases (described in section II) which were followed up for one year or more after removal of the foreign body. These cases are combined in table 3, which gives the results in 26 cases in which the foreign body had been in the eye from four months to twenty years before being removed, the cases then being followed up from one week to eight and one-third

years. Since 62.5 per cent of the first group and 80 per cent of the second group had less than 20/200 vision at the time of discharge, no serious error is introduced by combining the two groups of figures in order to obtain a comprehensive view of this type of case.

It is apparent from a study of these 26 cases that the continued presence of an intra-ocular foreign body for four months or more leads to definitely poor visual results even though the foreign body is subsequently removed. In the 3 cases in which vision of 20/20 was achieved, the foreign body was in the lens in 2 cases and in the vitreous in the other case. The foreign body was steel in each case and was removed eighteen months, five months and nine months, respectively, after the

TABLE 3.—*Foreign Body in Eye Four Months or More Before Removal* *

		Vision					Additional Data						
	Total	Less Than 20/200	20/200	20/70	20/20	Not Stated	Cataract	Aphacia	Secondary Membrane	Detachment	Vitreous Exudate	Siderosis	Scleral Wound
Anterior chamber	1	1	..	..	..	..	..	1	..	..	..	..	..
Ciliary body	4	3	..	1	..	..	1	1	..	1	1	2	1
Lens	7	2	1	..	2	2	1	6	..	..	..	1	..
Vitreous	5	4	..	..	1	..	2	1	..	2	..	3	1
Retina	3	2	1	..	..	..	1	1	..	1	..	..	..
Choroid	3	3	..	..	..	..	1	2	..	2	..	2	..
Unknown	3	3	..	..	..	..	1	2	1	1	..	1	..
Total	26	18	2	1	3	2	7	14	1	7	1	9	2
Percentage		69.2	7.7	3.9	11.5	7.7							
Percentage in surviving eyes (same)													

* One detachment was preceded by a scleral incision; (vitreous exudate was present in the same case).

primary injury. No siderosis was seen in these 3 cases. (It is a matter of common knowledge that the continued existence of a foreign body in the lens will result in less permanent damage to the eye than when the foreign body is in any other tissue of the eye). In the patient attaining 20/70 vision, the foreign body was steel and was removed from the ciliary body two and one-half years after the original injury. The case was then followed up for two and one-third years more, the vision being unchanged during this time. There was accompanying siderosis, with posterior synechiae and an immature cataract.

It might be noted that 9 of the 16 cases of siderosis are found in table 3, offering a partial explanation of the poor visual results.

IV. CASES IN WHICH THE FOREIGN BODY WAS NOT REMOVED

These cases fall into two groups: (*a*) those followed up less than four months after the original injury, and (*b*) those followed up for four months or more after the injury. Four months were taken as the

critical period because most of the enucleations should occur in the first group, while in the second group sufficient degenerative changes should have occurred to result in a definite loss of vision.

(*a*) There were 21 cases followed up less than four months, which may be briefly tabulated as follows:

Anterior chamber—1 (enucleated)
Iris—1 (20/100)
Ciliary body—3 (enucleated, 2; 20/50 and detachment, 1)
Vitreous—14 (enucleated, 12; 20/200, 2)
Unknown—2 (enucleated, 1; vision less than 20/200, 1)

Sixteen eyes (76.1 per cent) were enucleated at varying intervals of from five days to six and one-half weeks after the injury. The single detachment followed attempted removal through a scleral incision. The patient having vision of 20/100 was suspected of malingering; 1 patient with vision of 20/200 had a cataract, while the pathologic changes were not noted in the other case. In the remaining case (vision less than 20/200) there was a mature cataract.

(*b*) There were 21 cases which were followed up four months or more; they may briefly be tabulated as follows:

Ciliary body—3 (enucleated, 2; 20/200, 1)
Lens—2 (enucleated, 1; 20/70, 1)
Vitreous—6 (enucleated, 1; no vision, 1; vision less than 20/200, 1; 20/100, 1; 20/70, 1; 20/30, 1; 20/20, 1)
Retina—3 (vision less than 20/200, 1; 20/70, 1; 20/30, 1)
Choroid—4 (enucleated, 3; vision less than 20/200, 1)
Unknown—3 (enucleated, 2; vision less than 20/200, 1)

There were 3 detachments, 2 of which were preceded by a scleral incision, and 5 cases of siderosis.

The 8 enucleations (38.1 per cent) were done four months (2 cases), six months, seventeen months and nine, ten, thirteen and twenty years after the original injury. Of the remaining 13 cases, 6 were followed up for from six to twelve months, 4 were followed up for from one and one-half to four and one-half years, and the remaining 3 were followed up five and one-half, twelve and one-half and twenty-six years.

The patient with vision of 20/20 had a nonmagnetic foreign body in the vitreous. This case was followed up for seven months.

There were 2 patients with vision of 20/30. In the first, a steel particle 7 by 1 mm. was in the vitreous. Extraction of a cataract and de Wecker discission were performed within five and one-half months. When last seen (five and one-half months after the injury) two roentgenograms revealed nothing abnormal. The other patient attaining vision of 20/30 was a man who was seen immediately after being struck

in the eye by a flying particle. When first seen, vision was 20/20. There was no perforating wound of the globe, but examination of the fundus revealed a small foreign body (3 by 2 mm. in the roentgenogram) in the retina, surrounded by much pigment. The history of an injury five years earlier was then elicited. The vision fell rapidly to 20/200 owing to the development of a vitreous exudate just behind the lens. After intensive general treatment with sweats, mercury and injections of milk, most of this exudate cleared up during the next three weeks, and vision improved to 20/40. Vision was 20/30 six months after the second injury. This case demonstrated clearly that the presence of an intra-ocular foreign body renders the eye peculiarly vulnerable to what would ordinarily be a trivial injury.

TACLE 4.—*Foreign Body Not Removed* *

			Vision						Additional Data						
	Total	Enucleated	None	Less Than 20/200	20/200 to 20/100	20/70 to 20/50	20/30	20/20	Cataract	Aphacia	Secondary Membrane	Detachment	Vitreous Exudate	Siderosis	Scleral Wound
Anterior chamber	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..
Iris	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..
Ciliary body	6	4	..	..	1	1	..	..	..	..	..	1	..	1	1
Lens	2	1	..	..	..	1	..	..	..	..	..	..	..	..	1
Vitreous	20	12	1	1	3	1	1	1	2	4	..	1	1	1	3
Retina	3	..	..	1	..	1	1	..	..	1	1	..	..	..	..
Choroid	4	3	..	1	..	..	..	..	..	..	..	2	..	2	1
Unknown	5	3	..	2	..	..	..	..	1	1	1	..	..	1	..
Total	42	24	1	5	5	4	2	1	3	6	2	4	1	5	6
Percentage		51.7	2.4	11.9	11.9	9.5	4.8	2.4							
Percentage of surviving eyes (18)			5.6	27.8	27.8	22.2	11.1	5.6							

* Three detachments were preceded by a scleral incision (or wound). The foreign body was absorbed in 4 cases.

The foreign body was absorbed in 4 cases.

1. This patient was seen twenty years after injury. Vision was nil; the cornea was opaque, and there was lowered tension. The eye was enucleated. The pathologic report was as follows: There were a siderotic patch in the ciliary processes, retinal pigmentation and a calcareous lens; no foreign body was present. This is case 1 in table 5.

2. This patient was previously described as having vision of 20/30 (aphacia). A roentgenogram showed nothing abnormal after five and one-half months.

3. A nonmagnetic metallic foreign body entered the vitreous. There was a traumatic cataract. Linear extraction was made seven days after the injury for secondary glaucoma (a swollen lens). Roentgen examination gave negative results one month after injury. There were massive vitreous opacities which cleared in part, so that, nine months after the injury, vision was 20/70 (aphacia), and there was a great deal of connective tissue in the fundus.

4. This patient was seen fifteen months after the foreign body entered the eye. There was no history of removal. There were a swollen cataract and siderosis of the lens and iris. Roentgen examination gave negative results. Simple extraction was performed. Three months later, vision was limited to the ability to see movements of the hands (dense secondary membrane). This is case 15 in table 5.

Table 4 contains the visual results in the 42 cases in which the foreign body was not removed from the eye. If the visual results are calculated in percentages of the surviving eyes (18) and compared with those in the 26 cases in table 3, in which the foreign body was in the eye for four months or more before being removed, it is apparent that there are no striking differences between the results in the two types of cases, especially since both series are relatively short.

In table 4, 55.6 per cent of the patients attained vision of from ability to see movements of the hands to 20/100; 22.2 per cent had vision of 20/70 or 20/50, and 16.7 per cent had vision of 20/30 or 20/20.

In table 3, 76.9 per cent had vision of from ability to see movements of the hands to 20/200; 3.9 per cent had vision of 20/70, and 11.5 per cent had vision of 20/20.

In general, it would appear from the foregoing figures that if a foreign body remains in the eye four months or more, vision will probably be slightly better if the foreign body is not removed. There are, of course, other reasons which may demand that an attempt be made to remove the foreign body.

V. RELATION BETWEEN SCLERAL WOUNDS, DETACHMENT OF THE RETINA AND VITREOUS EXUDATE

Under the foregoing heading are included all types of scleral wounds, such as:

(*a*) Operative, for removal of the foreign body in the posterior segment.
(*b*) Accidental, when the foreign body entered through the sclera and was removed through the same wound (enlarged, if necessary).
(*c*) Cases in which the foreign body passed through the sclera twice on its way to the orbit.
(*d*) Cases which showed a scleral wound but in which the detachment was not discovered until the eye was sectioned after enucleation.

In 1 case of scleral incision (operative) a detachment was present before operation. This detachment was not considered as having resulted from the scleral incision.

It was felt justifiable to include accidental and operative scleral wounds together because there were: 25 operative scleral wounds, 7 (28 per cent) of which were followed by detachment, and 11 accidental scleral wounds, 4 (36.3 per cent) of which were followed by a detachment.

The preceding tables (1 to 4) give the number of scleral incisions, detachments and vitreous exudates which occurred in each group. If the total is taken for all groups, it is evident that in the 270 cases of this report, there were: 36 scleral wounds, 11 of which were followed by detachment (30.6 per cent), and 22 detachments, 11 of which were preceded by a scleral wound (50 per cent).

In 234 cases in which there was no scleral wound, there were 11 detachments (4.7 per cent).

With regard to vitreous exudate, there were: 36 scleral wounds, 10 of which were followed by vitreous exudate (27.8 per cent), and 24 cases of vitreous exudate, 10 of which were preceded by a scleral wound (41.7 per cent).

In 234 cases in which there was no scleral wound, there were 14 vitreous exudates (6.2 per cent).

As 2 of the vitreous exudates occurred in cases in which a detachment had also resulted from a scleral incision, it is apparent that 19 of the 36 scleral wounds were complicated by either a detachment, or a vitreous exudate or both, an incidence of 52.8 per cent of unfavorable sequelae.

Of the 11 detachments which were preceded by a scleral incision, 2 healed, with resulting vision of 20/20 (followed up for three months and four years, respectively).

Of the 11 detachments which were not preceded by a scleral incision, 1 healed, with a resulting vision of 20/70 (cataract, followed up for five months).

It cannot be definitely stated from these figures that absolutely uncomplicated cases would result in as many detachments if the foreign body were removed by the posterior route, because this method was used only in those cases in which there was a scleral wound (accidental) and in those in which the foreign body could not be brought into the anterior chamber. However, it does seem that the probability of a detachment occurring is definitely increased when removal through the sclera is either accomplished or attempted.

VI. SIDEROSIS

Among the 270 cases analyzed in this survey, there were 16 cases of siderosis (5.9 per cent). As most of these cases were seen at the Herman Knapp Memorial Eye Hospital from two months to twenty years after the original injury, siderosis was present in most of them when they were first seen; so these figures are of little value in determining the time of its onset. It was actually seen to develop only in case 7 (table 5) and possibly in case 2. Case 16 is noteworthy because

TABLE 5.—*Cases of Siderosis* *

Case	Seen After Injury	Findings at First Visit	Location of Foreign Body	Treatment	Vision at Last Visit	Followed After First Visit
1	20 yrs.	Vision, 0; opaque cornea; eye soft; siderosis of ciliary body (pathologic diagnosis)	Ciliary body	Enucleated; foreign body absorbed	Anophthalmos	No return
2	2½ yrs.	20/70; posterior synechiae; siderosis	Ciliary body	Magnet	20/70; immature cataract	2½ years
3	1¼ yrs.	20/70; diffuse siderosis	Ciliary body	Magnet	3/200; poor fundus reflex	1½ years
4	1 7/12 yrs.	Perception of light; faulty projection; cataract; siderosis of lens and iris	Lens	Magnet	Perception of light; faulty projection; cataract	5 months
5	8 mos.	Vision limited to ability to see movement of hands; traumatic cataract; siderosis of lens	Vitreous	Magnet	Perception of light; 20/30 after extraction of cataract; then a detached retina developed	1 year
6	9 mos.	20/50. p. c. d.; media hazy; siderosis of iris	Vitreous	Magnet	7/200; vision improved to 20/25, then decreased owing to exudate on lens	4 years
7	6 days	2/200; cataract; iridocyclitis	Vitreous	Not removable	20/100; siderosis after 5 months; foreign body encapsulated at 3½ years; cataract absorbed	4½ years
8	1½ yrs.	20/100; fundus indistinct; siderosis of iris and lens	Vitreous	Magnet	3/200; exudate in anterior chamber	2 months
9	2 mos.	20/70; media hazy; siderosis of iris	Vitreous	Magnet	20/20; no details	1¼ years
10	4 mos.	Vision limited to ability to see movement of hands; cataract; siderosis of iris	Choroid	Magnet	Ability to see movement of hands; aphacia; good opening	1½ years
11	9 yrs.	Vision, 0; deep anterior chamber; cataract; siderosis of iris	Choroid	Enucleated	Anophthalmos	7 years
12	7 mos.	20/100; media hazy; siderosis of iris	Choroid	Not removable	15/200; detachment of retina 2 months after first visit	5 months
13	10 mos.	20/70; cataract; siderosis	Choroid	Magnet	Perception of light; detachment occured 2 months after removal of foreign body; aphacia	1½ years
14	1¼ yrs.	Vision limited to ability to see movement of hands; cataract; siderosis; foreign body removed elsewhere 3 months previously	Unknown	Extraction of cataract	Ability to see movement of hands; thick secondary membrane	3 months
15	1½ yrs.	Perception of light; cataract; siderosis of iris; foreign body absorbed (roentgenogram showed nothing abnormal)	Unknown	Extraction of cataract	Ability to see movement of hands; thick secondary membrane	2 weeks
16	7 wks.	Perception of light; iridocyclitis; siderosis of iris	Posterior sclera and orbit	Enucleated	Anophthalmos	1⅚ years

* The foreign body was removed or the eye enucleated when the patient was first seen in all cases except case 3, in which the foreign body was removed one month after the patient was first seen.

siderosis of the iris was first seen seven weeks after the injury (iridocyclitis was also present). When the eye was enucleated, the foreign body was found in the sclera, extending into the orbit. (This is also case 11, table 6.)

In 5 cases (cases 1, 7, 11, 12 and 15), the foreign body was not removed. In case 1, it had absorbed; the eye was enucleated. In case 15 it had also absorbed, and the final vision was limited to ability to see movements of the hands (secondary membrane). In case 7 the final vision was 20/100 (aphacia); in case 12 it was 15/200 (detachment), and in case 11 the eye was enucleated.

In 1 case (case 16) the eye was enucleated when the patient was first seen because of iridocyclitis.

In the remaining 10 cases, the foreign body was removed two, four and one-fourth, eight, nine, ten, twelve, sixteen, eighteen, nineteen and thirty months, respectively, after the injury, with corresponding vision of 20/20, ability to see movements of the hands, perception of light, 7/200, perception of light, ability to see movements of the hands, 3/200, 3/200, perception of light and 20/70, after being followed up for one and one-fourth, one and one-third, one, four and one and one-third years, three months, one year, two months, five months and two and one-third years, respectively.

The visual results in the siderotic eyes were as follows:

Anophthalmos	—3 (18.6 per cent).....	Surviving Eyes
Vision <20/200	—10 (62.5 per cent).....	76.9 per cent
Vision =20/100	—1 (6.3 per cent).....	7.7 per cent
Vision =20/70	—1 (6.3 per cent).....	7.7 per cent
Vision =20/70	—1 (6.3 per cent).....	7.7 per cent

There were 3 detachments (2 followed the extensive use of a magnet).

In all cases except case 9, in which vision of 20/20 was obtained (the foreign body was removed two months after the accident) and maintained for more than one year, the presence of siderosis could be taken as an indication that degenerative changes had already begun in the intra-ocular tissues. Thus, in case 3, vision decreased from 20/70 to 3/200 in one year. In case 5, a temporary improvement to 20/30 was followed by a detachment. In case 6, vision improved from 20/50 to 20/25 two months after removal of the foreign body and then fell to 7/200 during the next ten months owing to the development of a pupillary exudate. In case 8, vision fell from 20/100 to 3/200 owing to a pupillary exudate. In case 12, attempted removal by the magnet was followed by a detachment two and one-half months later, and in case 13, vision fell from 20/70 to ability to see movements of the hands

TABLE 6.—*Foreign Body in Orbit (Double Perforation of Globe)* *

Case	Foreign Body; Size, Mm.	Seen After Injury	Examination at First Visit	Course	Final Result	Followed After Injury
1	Lead BB shot	4 days	Perception of light (faulty) eye soft; no red reflex; foreign body entered through sclera	Exudate and recurrent hemorrhages developed in vitreous; enucleated after 8 months for iridocyclitis; foreign body adherent to sclera	Anophthalmos	11 months
2	Steel 6 by 3.5	6 hours	Vision, 0; eye soft; corneoscleral wound; no reflex	Enucleated after 3 days; foreign body in Tenon's capsule	Anophthalmos	2 months
3	Steel 4.5 by 1.2	6 hours	20/200; corneoscleral wound; cataract nasally	Enucleated after 4 days for beginning panophthalmitis	Anophthalmos	No return
4	Steel 2.5 by 1.7	1 day	20/40; foreign body entered and left through sclera; shallow detachment	Vision, 20/70 8 days later; detachment healed in 3 to 6 weeks, with vision of 20/20	20/20	3 months
5	Lead 4 by 1.7	1 year	Vision, 0; phthisis; sympathetic ophthalmia in other eye (vision, 20/200)	Enucleated at once; vision in remaining eye 20/50 2 months later; then failed to light perception	Anophthalmos	3½ years
6	Steel (?)	8 days	Vision limited to ability to see movements of hands; traumatic cataract	Linear extraction 7 weeks after injury	No record	9 weeks
7	Steel 1 by 1	4 days	20/50; posterior cortical cataract; foreign body in posterior sclera	Foreign body removed 4 days later by anterior route; 5 weeks later, detachment	Ability to see hand movements; detachment	4½ months
8	Steel 5 by 2	6 weeks	1/200; cataract; foreign body entered and left through sclera	Shallow detachment and hemorrhage in vitreous seen 2 weeks later	Ability to see hand movements; detachment	2½ months
9	Steel 2 by 1	3 months	4/200; cataract; foreign body in posterior sclera	Foreign body removed at once by anterior route; cataract extraction 8 days later; hypopyon and vitreous exudate, 10 weeks later	Vision, 0; phthisis	8 months
10	Steel (?)	1 day	Vision, 0; beginning panophthalmitis	Enucleated 2 days after injury	Anophthalmos	3 years
11	Steel 11.5 by 1.5	7 weeks	Perception of light; wound at limbus; siderosis; cataract	Enucleated 1 week later; foreign body in posterior sclera and orbit	Anophthalmos	1⅝ years
12	Steel 5 by 3 by 1	6 hours	Vision limited to ability to see movements of hands; eye soft; wound of cornea; lens clear; subconjunctival hemorrhage nasally	Foreign body removed from under nasal conjunctiva at once; vision, 1/200 with organized vitreous exudate 1½ years later; lens clear	1/200	1½ years
13	Lead 4 by 1.7	6 hours	Perception of light; traumatic cataract; severe uveitis	Cataract absorbed; 2 years later, vision was limited to ability to see movements of the hands; vitreous exudate	Ability to see hand movements	2 years
14	Lead 2.5 by 2.5	3 days	1/200; lens dislocated; huge vitreous hemorrhage	Connective tissue in vitreous	Perception of light	20 years

* The exudate and hemorrhage in the vitreous in case 1 have not been included in the tables. The vitreous exudate in case 9 was not included in the tables because it followed the extraction of the cataract.

owing to the development of a detachment. Or among the 13 surviving eyes, vision improved in 2 cases, was unchanged in 5 cases, and became worse in 6 cases.

The foregoing facts bear out Verhoeff's contention that siderotic degeneration continues notwithstanding removal of the foreign body.

VII. FOREIGN BODY IN THE ORBIT (DOUBLE PERFORATION)

While these cases should theoretically attain ideal results as the foreign body is in the eye only for an instant, a study of 14 cases (table 6) demonstrates the exact opposite.

Apparently, the following four factors are of major importance:

(*a*) A double perforation leads to the escape of intra-ocular fluids at two points. These points are usually large enough to delay healing sufficiently long to allow definite hypotony to occur.

(*b*) Injury at two points leads to the possibility of hemorrhage at two points, with a resulting vitreous hemorrhage (Samuels [8])

(*c*) In general, for a foreign body to penetrate the globe twice, it must have a certain size and a certain velocity, since $E = \frac{1}{2}mv^2$, as pointed out by Parsons [9] in 1916.

(*d*) If retinal detachment tends to develop after a hole in the retina accompanied by changes in the vitreous, detachment should not be infrequent in these cases.

In this series (table 6) of 14 cases, I have included 3 cases in which the foreign body stopped in the posterior sclera, being partially in the orbit. There were 10 steel and 4 lead foreign bodies. Nearly all of them were moderately large, averaging 4.4 by 1.9 mm., while in 144 other cases, the average size was 2.6 by 1.2 mm.

There were 6 enucleations, 4 of which were done immediately, 1 after eight months and 1 after one year (the last-mentioned one was the case of sympathetic ophthalmia described in section II).

Of the 8 remaining eyes, vision was 20/20 in 1 (the healed detachment noted in the following paragraph), 1/200 or less in 6 and not stated in the last case.

There were 3 detachments (21.4 per cent of the total number of cases, or 37.5 per cent of the surviving eyes), 1 of which healed about six weeks after the injury, with vision of 20/20 for six more weeks, and there were 4 cases of vitreous exudate.

Of interest are cases 7 and 9. In both, the foreign body, which was apparently just through the posterior sclera, was brought forward into the anterior chamber and removed. A detachment developed in the first case, and phthisis in the second.

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8. Samuels, Bernard: Demonstration of Microscopic Preparations of Eyes Containing Foreign Bodies, Arch. Ophth. **7**:474 (March) 1932.

9. Parsons, J. H.: Tr. Ophth. Soc. U. Kingdom **36**:70, 1916.

In case 12 (Friedman [10]), the diagnosis of double perforation was made from the presence of a subconjunctival hemorrhage nasally, the foreign body having entered the eye through the cornea.

The impressive feature is that all of these patients did poorly with regard to enucleations, detachments and visual results. Even the case in which vision was 20/20 cannot conclusively be stated as exceptional, as it was followed up for only three months. Whether or not vision failed later is unknown.

VIII. CHANGES IN VISION OVER A PERIOD OF TIME

Several cases have shown remarkable changes in vision over a period of a year or less. Cases in which vision suddenly improved after removal of a cataract are not included here. All intervals of time mentioned are from time of removal of the foreign body.

(a) Glass, 4 by 2 by 2 mm., was present in the ciliary body (Vogt bone-free roentgenogram). It was removed by forceps ten days after injury; vision was limited to perception of light. One month later, vision was 20/100; two months later, 20/40; five months later, 20/30, and two and one-fourth years later, no change.

(*b*) A large (?) piece of steel was present in the vitreous. The patient was seen ten days after the accident; vision was 2/200; there were posterior corneal deposits; a hypopyon was present. The foreign body was removed. Two and one-half weeks later, vision was 20/200; one and one-half months later, 20/200, and from three to six months later, 20/50; there was still some exudate in the vitreous.

(*c*) There was a piece of steel in the vitreous. Hemorrhage was present in the vitreous; vision was 15/200. The foreign body was removed two days after injury. Twelve days later, vision was 18/200; there were a vitreous exudate and detachment in the temporal part of the retina. One month later, vision was 6/200; two months later, 10/200; three months later, 20/100 and the detachment was healed; five months later, 20/70; the field was good, and there was a capsular cataract.

(*d*) There was a piece of steel in the vitreous; a traumatic cataract was present; vision was 5/200. The foreign body was removed one day after injury. Six months later, combined extraction was performed. Seven months later, vision was 20/70 (vitreous hazy); eight months later, 20/50; nine months later, 20/25, and ten and one-half months later, 20/30.

(*e*) There was a piece of steel in the vitreous; vision was limited to the ability to see movements of the hands; the eye was red. The foreign body was removed two and one-half weeks after the accident. Five weeks later, vision was 4/200; nine weeks later, 5/200; ten weeks later, 20/70, and three months later, 20/70. (there was a corneal opacity with mixed astigmatism).

(*f*) There was a piece of steel in the choroid; lenticular and vitreous opacities were present; vision was 5/200. The foreign body was removed five days after the accident. Two weeks later, vision was limited to ability to see movements of the hands; six months later, 1/200, with a massive vitreous exudate; nine

10. Friedman, Benjamin: Subconjunctival Hemorrhage in Intra-Ocular Foreign Bodies, Arch. Ophth. **6**:919 (Dec.) 1931.

months later, 5/200; eleven months later, 20/50; one and one-half to one and three-fourths years later, 20/40, and one year and ten months later, 20/50; there were a posterior cortical cataract and some remaining vitreous exudate.

(*g*) There were a piece of steel, location not stated, and a traumatic cataract. The foreign body was removed six months after injury. The patient was first seen at the Herman Knapp Memorial Eye Hospital two and one-half years after removal of the foreign body; vision was 2/200; a cataract was present. Discission was performed. From three to five and one-half years after removal of the foreign body, vision varied from 20/30 to 20/40. The patient was struck in the eye eight years after removal of the foreign body; detachment occurred; vision was limited to the ability to see movements of the hands.

(*h*) A piece of steel was present in the vitreous for eight months; vision was limited to the ability to see movements of the hands; a traumatic cataract and siderosis were present. The foreign body was removed at once, and extraction of the cataract was performed one month later. Vision was 20/30 for five months; detachment then developed, which was unchanged one year after removal of the foreign body. (This is case 5, table 5.)

(*i*) A piece of steel was present in the vitreous for nine months; vision was 20/50; there were posterior corneal deposits; the media were cloudy; there was siderosis of the iris. The foreign body was removed. Vision improved to 20/20 two weeks after removal of the foreign body; two months later it was 20/25; one year later it was 7/200 owing to a capsular cataract (an exudate was present on the lens).

From a consideration of the foregoing cases, which were seen at regular intervals after removal of the foreign body, it is apparent that in all cases except *g* visual changes had become constant within a year from the time of removal of the foreign body. Case *g* scarcely belongs here because a second trauma was received by the eye.

Therefore, it is felt that there is some justification for taking one year as the minimum time of follow-up after removal of an intra-ocular foreign body.

IX. SUMMARY

In table 1 are presented 159 cases in which the foreign body was removed within four months and the cases were then followed up for from one week to ten and one-half months. Thirty-eight eyes were enucleated. Of the surviving 121 eyes, 19 per cent had 20/20 vision, 9.1 per cent had 20/30 vision, and 9.9 per cent had 20/40 vision.

In table 2 are presented 43 cases in which the foreign body was removed within four months and the cases were then followed up for from one to twenty years. One eye was enucleated. Of the surviving 42 eyes, 28.6 per cent had 20/20 vision, 16.7 per cent had 20/30 vision, and 7 per cent had 20/40 vision.

In table 3 are presented 26 cases in which the foreign body was in the eye from four months to twenty years before removal. There were no enucleations. In 11.5 per cent vision was 20/20. No other cases showed better than 20/70 vision, and in 69.2 per cent vision was less than 20/200. There were 9 cases of siderosis.

In table 4 are presented 42 cases in which the foreign body was not removed. There were 24 enucleations. Of the remaining 18 eyes, 5.6 per cent had 20/20 vision, 11.1 per cent had 20/30, and none had 20/40 vision. There were 5 cases of siderois. The number of enucleations was highest in this group, since these eyes were removed either because the foreign body was irremovable and there was chronic iridocyclitis, or because the eyes were badly damaged.

With regard to scleral wounds, detachment of the retina and vitreous exudate (or hemorrhage), 30.6 per cent of 36 scleral wounds were followed by detachment, and 27.8 per cent of 36 scleral wounds were followed by vitreous exudate. Since in 2 of the foregoing cases both a detachment and a vitreous exudate occurred following a scleral incision, 52.8 per cent of the scleral wounds were followed by detachment, exudate or both.

The incidence of detachment not preceded by scleral incision was 4.7 per cent, and the incidence of vitreous exudate not preceded by scleral incision was 6.2 per cent (these figures are based on 234 cases in which there was no scleral wound).

The value of the foregoing figures is not vitiated by the fact that 3 of the 22 detachments healed.

Siderosis was found in 16 (5.9 per cent) of the 270 cases (table 5). Three of the eyes were enucleated. Of the 13 surviving eyes, 7.7 per cent attained 20/20, 20/70 and 20/200 vision (1 case of each), and 76.9 per cent had vision of less than 20/200. In 6 of the 13 surviving eyes, vision definitely decreased after the foreign body was removed (the decrease was due to a detachment in 3 cases).

The 14 patients with double perforation (table 6) did poorly. Six eyes were enucleated. Of the 8 surviving eyes, 1 had 20/20 vision (healed detachment), 6 had vision of 1/200 or less, and vision was not reported in the remaining case. There were 3 detachments and 4 cases of vitreous exudate.

In section VIII, 9 cases are briefly abstracted in which vision changed remarkably over a period of time but tended to remain unchanged after from three to twelve months.

There were 2 cases of sympathetic ophthalmia among the 270 cases studied, both of which have been described in earlier sections of this paper. It is of interest that there were no cases of sympathetic ophthalmia among the 42 cases in which the foreign body was not removed, nor were there any among the 26 cases in which a foreign body was in the eye for from four months to twenty years before removal.

X. CONCLUSIONS

Although the 43 cases in which the foreign body was removed within four months and the cases then followed up for one year or more showed better visual results than the 159 cases similarly selected but

followed up for less than one year, it is felt that the 2 series are not strictly comparable, either as to the number or as to the distribution of cases (according to the site of the foreign body). It is my opinion that the only way to discover whether visual function tends to decrease after removal of a foreign body would be to compare the visual results in the same cases at periodic intervals. Only in this way can absolutely comparable statistics be obtained.

A scleral wound (operative or accidental) seems definitely to favor the development of a detached retina, a vitreous exudate or both. It should be used only when other methods of removing a foreign body fail.

If siderosis is present at the time a foreign body is removed, it is probable that vision will continue to deteriorate.

If a foreign body passes through the globe and stops in the posterior sclera or (and) orbit, the incidence of greatly lowered vision or enucleation will be greater than for a foreign body in any other location.

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CHEMISTRY OF THE LENS

V. RELATION OF THE ANATOMIC DISTRIBUTION OF THE LENTICULAR PROTEINS TO THEIR CHEMICAL COMPOSITION

ARLINGTON C. KRAUSE, M.D.
BALTIMORE

It has always been assumed, without actual evidence, that the various lenticular proteins were homogeneous in chemical composition. However, investigations on many other proteins have indicated that most proteins are heterogeneous in nature. On this basis, it is to be expected that the lenticular proteins are also composite. Technical difficulties prevent the fractionation of most of the naturally occurring proteins, but fortunately the nature of the anatomic growth of the lens aids in the isolation of the lenticular proteins, which are presumably of various ages. Since there is no reason to believe that the lenticular proteins of older cellular fibers are renewed during the life of the lens, the comparative quantitative analysis of the isolated proteins of the lamellae of the lens should give an insight into the process of growth of the lens.

The chronological changes in the lens protein also have a bearing on the etiology of senile and other forms of cataract. Cataract, in general, may be considered the result of injury to the lens. Since the proteins represent by far the major portion of the lens, and since their behavior in the injured lens varies with the age of the fibers, the study of the lamellar proteins adds indirectly to the pathologic history of cataract.

This paper is a report on the investigation of the quantitative anatomic distribution of the various lenticular proteins, and of the quantitative distribution of nitrogen in the lenticular proteins of the various concentric lamellae of the lens.

METHOD

The lenses were aseptically removed from normal fresh bovine eyes about one-half hour after the death of the animal. The substance of 200 lenses was separated from the capsule and weighed. To the lens substance sufficient distilled water was added to make 400 cc. of the mixture. The lens substance was then disintegrated by means of a mechanical stirrer. After the lenses lost one eighth of their weight, they were separated from the solution; 200 cc. of water was added, and the process was repeated until two fractions of about one-eighth, three of about one-fifth and one of about one-eighth the total amount of nitrogen of the lens were obtained. The proteins were estimated quantitatively in the various fractions according to the method previously described.[1] The mucoprotein was

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Krause, A. C.: Chemistry of the Lens: III. Autolysis of the Lenticular Proteins, Arch. Ophth. **10**:631 (Nov.) 1933.

determined in the following manner: A 100 cc. sample was boiled for fifteen minutes to coagulate the proteins. Sufficient sodium chloride was added to make a 5 per cent solution. After mixing, the coagulated protein was filtered off and again extracted with a 5 per cent solution of sodium chloride. To the combined filtrates, acetic acid was then added to give the maximum precipitation of mucoprotein. The precipitate was centrifugated off and washed with 95 per cent alcohol and then with ether. It was dried at 105 C. The amount of nitrogen was estimated by the Kjeldahl method. The mucoprotein gave a positive reaction to the tests for carbohydrate and sulphate after acid hydrolysis.

Two hundred lenses from 1 year old cattle were used for the determination of nucleic acid by the method of Levene.[2] The weight of nucleic acid corresponded to 0.07 per cent of the dry weight of the lens. Most of the nucleic acid was precipitated with the alpha crystallin in the ordinary method of preparation of this protein.

The proteins for chemical analysis were isolated as follows: The protein from about 2,000 bovine lenses, without capsules, was separated into lamellar fractions in the same manner as described. The solutions were diluted with water to contain approximately 0.5 Gm. of nitrogen per liter. The albuminoid was centrifugated off and then washed with water. To the supernatant solution, 10 per cent acetic acid was added until the maximal precipitation of alpha crystallin occurred. The insoluble protein was then centrifugated off. The beta crystallin was precipitated out of solution at 30 C. by adding magnesium sulphate until the solution was saturated. The precipitate was washed with a saturated solution of magnesium sulphate. The beta crystallin was then dissolved in water, coagulated by heat and washed with water until free from magnesium sulphate. After the removal of the beta crystallin, the filtrate, containing magnesium sulphate, was heated in order to coagulate the albumin. The coagula of albumin were removed and washed with water until free from magnesium sulphate. All of these protein preparations were washed with hot alcohol and cold ether to remove fatty substances and were dried in air. The procedure for the analysis, according to the method of Van Slyke and others, has been described in other papers.[3]

COMMENT

The anatomic distribution of the lenticular proteins shows that as the lens ages the albuminoid increases and the alpha crystallin decreases. The sum of the albuminoid and alpha crystallin nitrogen is approximately constant in all fractions except the last, which represents the remains of the nucleus more or less at an early fetal stage of development. The analysis suggests that alpha crystallin is converted (but not "denatured") into the inert albuminoid as the metabolic activity is diminished during the growth of the lens fiber. The beta crystallin remains practically constant in amount during the aging process. Pep-

2. Levene, P. A.: Darstellung und Analyse einiger Nucleinsäuren: VIII. Mitteilungen über die Milznucleinsäure, Ztschr. f. physiol. Chem. **45**:370, 1905.

3. Krause, A. C.: (*a*) Chemistry of the Lens: I. Composition of Albuminoid and Alpha Crystallin, Arch. Ophth. **8**:166 (Aug.) 1932; (*b*) II. Composition of Beta Crystallin, Albumin (Gamma Crystallin) and Capsule, ibid. **9**:617 (April) 1933.

tone and nonprotein nitrogen are probably the most active vital nitrogenous portions. The mucoprotein is the cementing substance of the lens fibers.

The distribution of the proteins in the 1 year old bovine whole lens, calculated in percentage from the nitrogen content of the individual proteins (table 1), is shown in table 2.

Mörner [4] evidently used old lenses, which naturally contained large amounts of albuminoid and small amounts of alpha crystallin. Moreover, he permitted his solution of beta crystallin to stand for several

TABLE 1.—*The Percentage of Distribution of Nitrogen in the Lenticular Layers of 100 Lenses (Age 1 Year)*

Lamellar Fractions (From Without Inward)	Nitrogen, per Cent						Average Percentage of Total Nitrogen of Whole Lens
	I	II	III	IV	V	VI	
Entire lens	14.7	12.3	18.5	19.6	21.5	13.4	
Albuminoid	0.4	4.8	7.0	9.9	13.2	38.4	11.87
Alpha crystallin	37.4	35.1	34.9	33.3	30.7	12.4	31.01
Beta crystallin	55.3	54.4	53.5	53.9	53.9	47.2	53.10
Albumin	2.3	2.4	1.5	1.2	1.1	0.9	1.50
Mucoprotein	1.3	1.1	1.1	0.4	0.3	0.4	0.72
Peptone	0.4	0.4	0.1	0.1	0.1	0.1	0.19
Nonprotein	2.9	2.8	1.9	1.2	0.7	0.6	0.60

TABLE 2.—*Distribution of Proteins*

Protein	Krause (Age 1 Year) per Cent	Mörner (Age Unknown) per Cent
Albuminoid	12.50	48.6
Alpha crystallin	31.74	19.4
Beta crystallin	53.39	31.4
Albumin	1.46	0.6
Mucoprotein	0.84	...
Nucleoprotein (as nucleic acid)	0.07	...

days, which evidently altered the physical properties of this labile protein.

The nitrogen distribution of the proteins definitely shows that the proteins are heterogeneous in composition. If the range of error of the Van Slyke method is taken into consideration, certain deductions may be made from the tables showing the variation of the nitrogen distribution with the age of the layers of the lens (tables 3, 4 and 5). The significant changes with age are given in table 6. The four lens proteins are therefore heterogeneous. There is no indication of the nature of the process of formation of albuminoid from alpha crystallin and of beta crystallin from albumin [3a] or vice versa.

4. Mörner, C. T.: Untersuchungen der Proteïnsubstanzen in den lichtbrechenden Medien des Auges, Ztschr. f. physiol. Chem. **18**:233, 1894.

TABLE 3.—*Nitrogen in the Albuminoid as Determined by the Method of Van Slyke*

Lamellar Fractions (From Without Inward)	Nitrogen, per Cent					
	I	II	III	IV	V	VI
Amide	5.49	3.96	4.89	4.80	3.70	4.97
Humin	1.44	1.84	1.70	1.45	1.09	1.05
Cystine	1.80	1.70	2.01	2.08	1.73	1.76
Arginine	27.11	20.61	20.60	20.97	19.06	18.27
Histidine	6.03	4.09	3.60	4.90	7.24	7.58
Lysine	5.36	7.85	6.80	7.63	9.27	9.60
Amino of filtrate	46.96	54.13	54.42	54.23	53.62	52.63
Nonamino of filtrate	5.89	7.00	5.00	4.30	4.34	3.71
Recovered	100.08	101.18	99.02	100.36	100.05	99.57

TABLE 4.—*Nitrogen in the Alpha Crystallin as Determined by the Method of Van Slyke*

Lamellar Fractions (From Without Inward)	Nitrogen, per Cent					
	I	II	III	IV	V	VI
Amide	3.71	3.31	3.23	3.43	4.66	4.61
Humin	0.88	0.96	1.10	1.36	1.01	0.99
Cystine	1.16	1.06	1.06	1.23	1.47	1.98
Arginine	17.63	17.55	19.15	19.05	18.75	19.20
Histidine	4.67	4.96	5.11	5.50	5.61	4.16
Lysine	8.86	10.40	9.36	7.53	6.64	6.92
Amino of filtrate	57.10	55.50	53.18	53.60	53.82	52.75
Nonamino of filtrate	5.28	4.62	7.10	8.32	8.76	10.55
Recovered	99.29	98.36	99.29	100.02	100.72	101.16

TABLE 5.—*Nitrogen in the Beta Crystallin and Albumin as Determined by the Method of Van Slyke*

	Nitrogen, per Cent					
	Beta Crystallin Fraction (Outer)		(Inner)	Albumin Fraction (Outer)		(Inner)
	I-II	III-IV	V-VI	I-II	III-IV	V-VI
Amide	6.03	6.01	7.20	8.80	7.51	7.72
Humin	1.24	1.12	1.18	1.32	0.55	0.75
Cystine	2.66	2.38	4.90	3.61	2.74	3.21
Arginine	16.60	18.52	18.66	16.10	18.21	21.44
Histidine	3.98	3.85	3.56	3.52	3.69	2.14
Lysine	6.22	6.01	4.70	3.52	5.46	7.50
Amino of filtrate	58.06	56.00	53.15	55.38	54.62	50.18
Nonamino of filtrate	6.62	5.96	7.34	7.32	7.30	7.50
Recovered	101.41	99.85	100.69	99.57	100.08	100.44

TABLE 6.—*Significant Changes in the Nitrogen Distribution with Age*

Nitrogen	Albuminoid	Alpha Crystallin	Beta Crystallin	Albumin
Arginine	Decreases	.....	.....	Increases
Lysine	Increases	Decreases	.....	Increases
Cystine	.....	.....	Increases	.....
Nonamino	Decreases	Increases	.....	.....
Amino	.....	.....	Decreases	Decreases

CONCLUSIONS

Albuminoid, alpha crystallin, beta crystallin and albumin are heterogeneous proteins, which vary in chemical composition during the growth of the lens. The anatomic distribution of the proteins suggests that albuminoid is derived from alpha crystallin, and that beta crystallin and albumin are interrelated chemically.

The material was obtained through the courtesy of Mr. R. L. Fox. Many of the technical procedures were performed by Mr. Warren Tauber.

HOLE IN THE MACULA

REPORT OF A CASE IN A PATIENT UNDER OBSERVATION

JOSEPH WILLIAM CRAWFORD, M.D.
SAN FRANCISCO

While a hole in the macula is not an exceedingly rare condition, few ophthalmologists have seen many of these lesions. Furthermore, most of the cases reported came under observation with the hole already fully developed. The events preceding the appearance of the lesion could be surmised only from the more or less indefinite history given by the patient. In not more than three cases thus far reported have ophthalmologists had the opportunity to see the lesion in its various stages of development. Having had such a case come under my observation, I wish to report a typical hole in the macula and the events leading to its formation.

Briefly, a hole in the macula has the following characteristics: Directly at the macula is seen a sharply circumscribed, circular or oval punched-out area, corresponding closely in size to the macular reflex seen in the normal eye, that is, about one-third to one-half disk diameter. Haab [1] suggested that the hole is always circular at first, but may become oval as the result of distortion by contraction of the underlying tissue. That this area is depressed below the level of the surrounding retina is proved by its parallactic movement when viewed ophthalmoscopically, and its depth can be estimated. The depth in the cases reported varied from 0.3 to 0.5 mm. Probably, as Ogilvie [2] suggested, the intermacular retinal area sinks to the level of the fovea.

The lesion is maroon or deep red, resembling the lesion seen at the macula in cases of embolism of the central retinal artery. The floor of the lesion is usually studded with tiny white or yellow spots, probably remnants of retinal tissue. Usually a few cholesterol crystals are seen on the floor. Immediately surrounding the hole may be seen a corona, which probably is caused by edema of the adjacent retina. Except for the lesion at the macula and a small ring of retina immediately sur-

Read before the Pacific Coast Oto-Ophthalmological Society, June 28, 1933.
From the Department of Surgery, Division of Ophthalmology, University of California Medical School.

1. Haab: Ztschr. f. Augenh. **3**:113, 1900.

2. Ogilvie, F. M.: On One of the Results of Concussion Injuries of the Eye ("Holes" at the Macula), Tr. Ophth. Soc. U. Kingdom **20**:202, 1900.

rounding it, the fundus is unaltered. The vision is usually reduced to 6/60, but it may be better if some of the nerve fibers of the macular bundle have escaped the destructive process. A central scotoma for form and color is found, usually corresponding in size and shape to the retinal lesion.

In reviewing the historical aspects of hole in the macula, one is struck by the fact that the lesion was seldom recognized before 1900. No nontraumatic case was reported prior to that time, and but three caused by trauma. In fact, the possibility of a cause other than trauma was not suspected until 1900, when Kuhnt [3] noted the appearance of the lesion in several patients in late middle life who disclaimed injury.

In 1869, Knapp [4] reported the first case of hole in the macula. This was followed in 1871, by Noyes' [5] report of a case in a child 13 years old. In 1889, Hartridge [6] reported a typical case in a man 23 years of age. All three cases were traumatic in origin.

In 1900, Kuhnt [3] reported four cases of macular hole. He named the condition "retinitis atrophicans sive rareficans centralis." Only one of the four patients had a history of preceding trauma; two were in late middle life. In the same year, Haab [1] reported twelve cases, in three of which there was no history of trauma. In 1900, Ogilvie [2] collected fifteen cases, all traumatic in origin; only one was in a person of advanced years.

De Schweinitz,[7] in 1904, described two cases occurring in his practice, one of which appeared in a patient under observation for an identical condition in the other eye. In this case, that of a woman 55 years old, there was a history of bilateral iritis following an attack of influenza, and before the hole appeared de Schweinitz noted an area of macular degeneration.

Clapp,[8] in 1913, reviewed the literature and summarized the hypotheses as to the causation of the lesion. Recently, Bedell [9] presented a series of photographic studies of the condition.

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3. Kuhnt: Ztschr. f. Augenh. **3**:105, 1900.

4. Knapp, H.: Ueber isolierte Zerreissungen der Aderhaut in Folge von Traumen auf dem Augapfel, Arch. f. Augen- u. Ohrenh. **1**:6, 1869.

5. Noyes, H. D.: Detachment of the Retina with Laceration at the Macula Lutea, Tr. Am. Ophth. Soc. **1**:128, 1887.

6. Hartridge, G.: Peculiar Appearance at Macula, Tr. Ophth. Soc. U. Kingdom **9**:144, 1889.

7. de Schweinitz, G. E.: Concerning Certain Non-Traumatic Perforations of the Macula Lutea, Tr. Am. Ophth. Soc. **10**:228, 1904.

8. Clapp, C. A.: Hole at the Macula, Ophth. Rec. **22**:79, 1913.

9. Bedell, A. J.: Photographic Study of Holes in Macular Region and Associated Changes, Am. J. Ophth. **10**:890, 1927.

The factors ordinarily ascribed to the causation of this lesion are as follows:

Traumatic.....	1. Contusions of the eyeball. 2. Penetration of the globe by a foreign body, with or without its retention.
Nontraumatic.....	3. Retinal vascular changes resulting from generalized vascular degeneration. 4. Rarefaction of tissue at the macula from inflammation or, more probably, from cystoid degeneration (Fuchs). 5. Inflammatory disease of the choroid or retina.

The mechanism of production in the traumatic cases is easily understood. The explanation for the appearance of the lesion in patients who give no history of trauma is more obscure. Kuhnt [3] was the first to recognize the nontraumatic type. He suggested that in many cases trauma was perhaps only a coincidental factor in the production of the lesion, and stated that none of the many cases of Berlin's edema, or ruptured choroid, previously reported had been accompanied by a hole in the macula. He saw a relationship between senile circulatory changes and the condition at the macula and was of the opinion that there was retinitis of the posterior pole of the eyeball, which tended to cause central atrophy of its tissues and the formation of a hole.

Haab [1] offered the suggestion that a hole in the macula might result from retinal arteriosclerosis, and might appear spontaneously in persons of advanced age. In one case, in which there was obvious generalized vascular degeneration, he offered the hypothesis that there was insufficiency of nourishment of the fovea, with resultant necrosis and formation of a hole.

De Schweinitz [7] believed the condition to be a degenerative process particularly affecting the neuro-epithelial layer of the retina and resulting in atrophy of the tissues.

Lister,[10] who, in 1924, thoroughly reviewed the entire subject of retinal holes, said that retinochoroidal degeneration was the most probable cause of nontraumatic holes.

Fuchs [11] and others considered cystoid degeneration a frequent cause of the condition. Such cysts frequently are found near the ora serrata, though rarely at the macula.

Elschnig's theory as to causation is that there is a combination of retinal detachment with a pegging-out of the retina to the choroid by preexisting areas of retinochoroiditis.

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10. Lister, William: Holes in the Retina and Their Clinical Significance, Brit. J. Ophth. **8**:1, 1924.

11. Fuchs: Text-Book of Ophthalmology, ed. 8, Philadelphia, J. B. Lippincott Company, 1924, p. 748.

Collins [12] expressed the belief that a hole in the macula can be explained by a subchoroidal hemorrhage, which by its pressure causes ischemia of the capillary plexus in the choroid. The retina at the macula, being almost entirely dependent for its nutrition on this underlying choriocapillaris, suffers from this cessation of circulation, and a hole results.

Coats [13] likewise expressed this opinion, basing his conclusion on a pathologic study of four cases, none of which were examined ophthalmoscopically before the eyes were enucleated. He stated that macular holes are the result of edema of the retina at the posterior pole of the eyeball. The edema is the result of a temporary ischemia of the choroid caused by a subchoroidal hemorrhage. He stated that in the absence of trauma the same effect can be produced by toxins in the vitreous resulting from iridocyclitis or from retinal vascular disease. Two of his cases were of the nontraumatic type.

That the macula is the area most likely to be affected by such a condition is understood easily when one considers the complex anatomy of the region. The edges of the macula really form a low wall, made up of the ganglion cell layer of the retina, which is raised above the level of the surrounding retina. Hence, the edges represent the thickest part of the retina, being from 0.275 to 0.41 mm. thick. The bottom of the fovea is the thinnest part, the inner layers of the retina being entirely absent here. It varies in thickness from 0.075 to 0.12 mm. (Salzmann [14]). In other words, the entire funnel-shaped depression of the fovea is formed at the expense of the retinal tissue. This cup is likewise devoid of retinal vessels, and the cerebral layers of the retina spread apart here, the nerve elements being almost entirely those of the first neuron, with a very few nerve fibers and a few isolated cells of the second neuron (Salzmann [14]). This anatomic arrangement, while a perfect one for functional activity, offers poor protection against assault.

REPORT OF CASE

Mrs. B., a housewife, aged 62, consulted me on Oct. 22, 1930, complaining of ocular symptoms explainable by a refractive error. Refraction brought her vision up to 1.2 for each eye, and except for retinal arteriosclerosis not inconsistent with her age, no ocular abnormalities were detected. Both macular areas were normal. The perimetric fields revealed no scotomas.

The patient returned on October 24 and was discharged on November 20, having been relieved of the symptoms of eyestrain by the wearing of her new

12. Collins, E. Treacher: Concussion Injuries of the Visual Apparatus, Tr. Ophth. Soc. U. Kingdom **37**:112, 1917.

13. Coats, George: The Pathology of Macular Holes, Roy. Lond. Ophth. Hosp. Rep. **17**:69, 1907.

14. Salzmann: Anatomy and Histology of the Human Eyeball, translated by E. V. L. Brown, Chicago, University of Chicago Press, 1912.

glasses. She was seen on Aug. 13, 1931, at which time vision was: right eye, 1.2; left eye, 1. At this time she did not complain of ocular symptoms. Examination revealed no change in the refractive error. The tension and the perimetric fields were normal. The left fundus, however, showed a very small area of chorioretinitis confined to the macular region. No hemorrhages were seen, and the affected area corresponded exactly in location to the lesion that appeared later, though it was smaller. The normal perimetric field and the vision of 1 are signifi-

Fig. 1.—Photograph of the fundus.

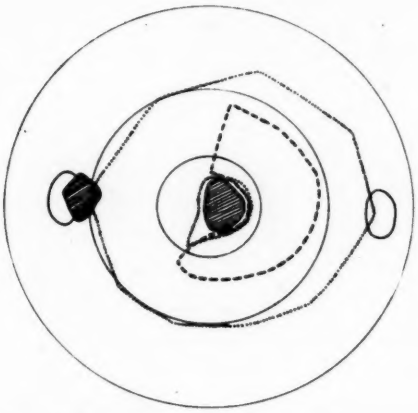

Fig. 2.—Perimetric field taken on Oct. 12, 1932 (objects, 6 mm.; radius, 1 meter). The dotted line indicates red; the broken line, green, and the areas of diagonal lines, form.

cant of the fact that the condition was not marked at this time. The patient was referred to her general physician and advised to return if any ocular symptoms appeared.

The patient returned on July 19, 1932, saying that the vision of the left eye had been failing gradually. She had no other ocular symptoms. Vision, with correction, was 1.2 in the right eye, and 0.1 in the left. Examination of the fundus revealed a typical hole in the macula. No hemorrhages were seen. No areas of chorioretinitis were seen in any part of the fundus. Since this visit the appearance of the fundus has not changed appreciably.

The accompanying photograph (fig. 1) shows the appearance of the fundus very well. The lesion is maroon and very slightly ovoid in the longitudinal diameter. The edges are clearcut. The floor of the lesion is depressed beneath the level of the surrounding retina. The amount of depression is difficult to measure, although it certainly cannot be more than 1 diopter. That the floor is definitely lower than the surrounding retina is determined easily by watching the parallactic displacement when the condition is viewed through a binocular ophthalmoscope. The floor is stippled, five small, irregular white spots being scattered over its surface. These may be remnants of retinal tissue, although they are too small to be differentiated. Two crystals, probably cholesterol, are present on the floor. A corona of pale retina surrounds the hole. This area is not elevated above the surrounding retina. The remainder of the fundus is normal except for moderately advanced arteriosclerosis. No hemorrhages are present.

The perimetric field (fig. 2) reveals a central scotoma for form and color corresponding in size and extent to the lesion seen with the ophthalmoscope. There has been no change in the condition since the lesion first appeared, the vision, perimetric fields, etc., remaining unchanged.

A physical examination was made by Dr. John Gallwey. The only positive findings were hypertension (blood pressure 196 systolic and 130 diastolic), mild secondary anemia and a faint trace of albumin in the urine. The cardiovascular condition was normal. No foci of infection were present, and all laboratory tests, including Wassermann and tuberculin tests, were negative.

COMMENT

Rarely have ophthalmologists had the opportunity to observe, step by step, the actual development of a hole in the macula in a previously healthy retina. Both Elschnig and de Schweinitz [7] reported cases in which the eyes were seen before the macular hole appeared, and in which a hole developed while the patient was under observation. These cases, like my own, were of the nontraumatic type. In the case reported by de Schweinitz,[7] an area of retinal degeneration at the macular area preceded the appearance of a hole, and in Elschnig's case an area of retinochoroiditis was first noticed at the macula.

My patient, at her first visit, had a normal macula; at the second, an area of chorioretinitis confined to the region of the fovea, and at the third, a hole at the macula. The patient is certain that at no time was there trauma that could have caused the lesion. The physical findings, except for generalized vascular changes not inconsistent with her age, were normal.

I believe that it is not inconsistent with the facts to assume that in this case the hole in the macula resulted from a localized degenerative process confined to the macular region, manifesting itself first as a small spot of circumscribed chorioretinitis. In view of the absence of any discernible foci that could have been responsible for an inflammatory process in the eye, and in the presence of hypertension and signs of retinal arteriolosclerosis, it is assumed that this retinal degen-

eration was but a part of the generalized vascular degeneration which was present.

The possibility that a preexisting cystoid degeneration of the retina acted as a causative factor in the production of this lesion must be considered. Cystoid degeneration of the retina is found so commonly in sections of the eyeball that it must be considered physiologic, at least in the peripheral portion of the retina. Though seen most commonly in senility, these cysts are often found in early adult life. It is impossible to say whether this area of the retina was already the site of cystoid degeneration, although the fact that the patient had vision of 1.2 in the eye only a year previously argues for a normal macular structure.

The chorioretinitis first seen in the macular area had no characteristics by which one could differentiate it from the same condition appearing frequently eleswhere in the fundus, especially in people of advanced years.

RODENT OR MOOREN'S ULCER OF THE CORNEA

REPORT OF THREE CASES WITH HEALING

SANFORD R. GIFFORD, M.D.
CHICAGO

The form of keratitis described by Mooren [1] in 1867 as chronic serpiginous or rodent ulcer may not be familiar to some ophthalmologists, as it is rare. Only seventy cases were found in a recent review of the literature by Heintz,[2] and he quoted only two reports by Americans, those of H. Gifford and Verhoeff. Bedell [3] recently quoted two typical cases, both resulting in loss of the eye. It is not so rare, however, but that most ophthalmologists of experience should see one or more cases during a lifetime. Heintz reported five typical cases from the Kiel clinic, and I have seen at least six.

Mooren's original description of the condition, which he first separated from the group of marginal ulcers, is as follows:

> The disease always begins at the margin of the cornea, progresses forward in irregular processes and does not stop, so far as my experience goes, until the whole corneal surface is involved. It is characteristic that a narrow gray line of infiltration, which changes continually with the progress of the disease, marks off the border of the uninvolved cornea. This line is at the same level as the healthy portion of the cornea and a little higher than the diseased portion, so that it always and under all circumstances appears to be undermined. While the uninvolved cornea preserves its transparence to the last moment, the development of a close superficial network of vessels begins on the diseased portion. In spite of the great pain and the rapid formation of new vessels, I have never seen hypopyon occur.

Perforation likewise never occurs spontaneosuly, though in one of my cases it had occurred following an injury to the diseased eye, with resulting phthisis bulbi. In the beginning, as described by Heintz, one or more deep gray infiltrates appear near the limbus and merge to form a shallow furrow. In this stage, the picture differs little from an ordinary marginal ulcer. The ulcer progresses toward the corneal center, however, extending also at both ends, eventually to surround the cornea. At the same time healing occurs at the periphery with the formation of

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From the Department of Ophthalmology, Northwestern University Medical School.

1. Mooren, Albert: Ophthalmologische Beobachtungen, Berlin, A. Hirschwald, 1867, p. 170; quoted by Schieck.[4]
2. Heintz, G.: Ztschr. f. Augenh. **77**:289, 1932.
3. Bedell, A. J.: Mooren's Ulcer, Arch. Ophth. **8**:782 (Nov.) 1932.

new vessels. Healing may appear complete for periods of days or weeks, but later the border extends farther centrally. Although the pain is usually severe, the congestion may be only moderate, with little or no accompanying iritis. Schieck [4] emphasized the fact that with the slit lamp the cornea just beyond the gray border shows fine opacities and yellowish foci of infiltration. H. Gifford [5] and others have called attention to the marked swelling and inflammation of the conjunctiva at the limbus, which is often raised in a red ring around the diseased area, and this is confirmed by Heintz, who found in sections of one case an active infiltration and loss of substance of the sclera in this area. It is this zone of scleritis and episcleritis which gives some early cases a certain resemblance to sclerosing keratitis, in which, however, ulceration of the cornea rarely if ever occurs.

The name rodent ulcer, while suggestive of the course of the disease, has perhaps proved confusing, as suggesting the entirely different rodent ulcer of the skin. The name chronic serpiginous ulcer is also misleading, since the only point of similarity to ordinary serpent ulcer is the undermined, advancing border. Mooren's ulcer requires months, usually from three to six months, to cover the cornea and, as previously stated, hypopyon and spontaneous perforation probably never occur. The histology of rodent ulcer, as shown by about eighteen cases, is that of a chronic infiltration, chiefly with round cells, of the anterior third or two thirds of the cornea. The loss of substance usually concerns the anterior third and is replaced by a thin layer of vascular connective tissue except near the advancing border, where the infiltration extends under the epithelium and into the stroma of the previously uninvolved cornea. Granulation tissue is present over the sclera beyond the limbus, and there may be loss of substance in the sclera itself.

The etiology of the disease is unknown. Three theories have been upheld. One insists on an infectious agent, although bacteriologic findings have been negative or have shown various organisms, presumably secondary invaders. A general state of malnutrition has been held responsible by some authors, although many cases have occurred in apparently healthy persons. A trophic disturbance due to primary pathologic changes in the fifth nerve has been maintained by Junius and others, as was suggested by the loss of sensitivity in the involved area. This exists in other forms of keratitis, however, and is considered by Heintz as probably secondary to damage of the nerves by the ulcerative process.

The prognosis in cases of rodent ulcer is usually considered bad. Schieck stated: "In view of our ignorance of the nature of the disease

4. Schieck, F.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 4, p. 347.

5. Gifford, H.: Klin. Monatsbl. f. Augenh. **37**:102, 1899.

and the tendency to relentless progress, it is a severe task to treat a patient with rodent ulcer. Usually one is in a hopeless battle." Usually, as stated, the cornea is completely covered, the resulting scar allowing very little vision, seldom over 20/200 and usually much less. In three of Heintz' five cases, the eye required enucleation because of pain. Since the disease frequently involves the second eye, the danger of practical blindness is great. The second eye was involved in three of my six cases and in one of Heintz' five cases.

On account of this gloomy prognosis, it seemed worth while to report three cases in which healing with good vision was obtained, apparently as the result of the therapeutic measures employed. One of these was previously reported in an article on delimiting keratotomy.[6] Photomicrographs of a typical case not seen clinically but studied in sections from a specimen sent me by Dr. L. W. Dean of Council Bluffs, Iowa, are included to illustrate the pathologic anatomy. They show the changes described as characteristic (fig. 1). The hypertrophy of the epithelium over the underminded border mentioned by some authors is especially well seen.

The treatment of Mooren's ulcer has, as a rule, been conservative, probably because of the unfavorable prognosis concurred in by most authors. Attempts to build up general resistance by injections of foreign proteins and other means have been employed, and Krückmann has reported a favorable result in which cod liver oil seemed to play a determining rôle. Krassó [7] recently reported a series of five cases which she considered abortive forms of Mooren's ulcer which healed rapidly under Bucky's border rays (*Grenzstrahlen*). The difference between her cases, which seem to have resembled severe catarrhal ulcers, and the typical picture of Mooren's ulcer is very great, and they could hardly be compared to those to be described. Krassó stated that roentgen therapy was employed with success by Salvatori, but that few details were given in his report which could confirm his diagnosis. Hence it cannot be said that irradiation therapy has proved its worth in Mooren's ulcer. In my case 3, phototherapy proved of only temporary benefit, and in case 1, of no benefit. Cauterization has often been employed, but usually without success, which, according to Schieck, is to be expected in a condition of trophic or degenerative nature. Attempts to produce hypotony, as was done in two of my cases, have rarely been reported. A. Fuchs [8] recently reported two cases in which this principle was employed with success. In one case a small incision was made in the advancing border, and this was reopened five times during fourteen days.

6. Gifford, S. R.: Am. J. Ophth. **5**:697, 1922.
7. Krassó, I.: Ztschr. f. Augenh. **77**:182, 1932.
8. Fuchs, A.: Brit. J. Ophth. **17**:193, 1933.

The ulcer began to heal at once, and vision of 5/8 was secured. In a second more advanced case peripheral paracentesis was performed and repeated fifteen times. Healing resulted, but vision was limited to the counting of fingers. These cases of Fuchs seem from his description to have been true Mooren's ulcer. He stated that Wibo employed puncture of the cornea with the actual cautery in Mooren's ulcer with success.

Fig. 1.—*A*, low power view of the anterior segment in Mooren's ulcer. *B*, higher magnification of the undermined, advancing border of the ulcer.

Nechitch [9] employed fistulization of the cornea in three typical cases, with excellent results. In order to make a large enough opening with the cautery, he first incised the ulcer with the keratome, inserted a broad spatula, which protected the lens, and applied the cautery to the cornea on the spatula. Such an opening remained patent for almost fifteen days.

9. Nechitch: Ann. d'ocul. **163**:268, 1926.

In one bilateral case sliding conjunctival flaps and other measures had failed, but healing was prompt after the puncture, with resulting vision of 3/10 and 6/10. In the other two cases final vision was 4/10 and 8/10.[10]

The use of a conjunctival flap has been attempted by a number of ophthalmologists, but as in a case that I observed this method has often failed. Kreiker[11] expressed the belief that failure occurs because the flap is made to cover only the obviously affected area, and after a few days it retracts and exposes the advancing border. He described three cases in which a flap was drawn from above to cover the cornea completely. Healing began at once in all three cases. After from two to three months a small opening was made in the conjunctiva over the unaffected cornea, following which the flap retracted around this and exposed the whole unaffected area. In no cases did the ulcer recur.

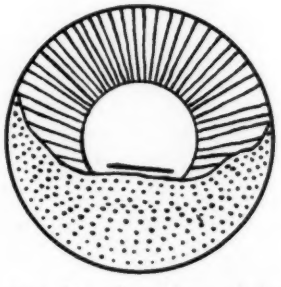

Fig. 2 (case 1).—Location of keratotomy.

Vision was 1/4 in one case, but considerably less in the other two, owing to the advanced stage at which the operation was performed.

REPORT OF CASES

CASE 1.—Fannie D., a colored woman, aged 52, appeared at the Northwestern University Clinic on Dec. 10, 1930, complaining of pain in the left eye for the past month. Vision was 10/100. A small marginal ulcer of the cornea was present at 7 o'clock. The ulcer improved somewhat after the instillation of atropine and applications of trichloracetic acid, but the pain and congestion continued, and in spite of applications of trichloracetic acid, mercurochrome and iodine twice a week and three treatments with the irradiation lamp, the opacity progressed until within one month it occupied the lower one half of the cornea from 3 to 9 o'clock. Most of the surface was smooth, only the central border and the two ends of the crescentic opacity showing active infiltration and staining with fluorescein. The patient was under competent treatment for syphilis, and the Wassermann reaction, which was 3 plus, became negative during treatment. Intradermal tuberculin tests gave

10. I was under the impression that Sondermann and Sondermann had reported a case in which healing occurred after trephining of the ulcer, but I could not find it on consulting the article (Klin. Monatsbl. f. Augenh. **88**:189, 1932).

11. Kreiker, A.: Klin. Monatsbl. f. Augenh. **90**:373, 1933.

negative results. The patient was thin and appeared undernourished. Cultures of the ulcer were negative, as was direct inoculation of a rabbit's cornea with material from the active border.

At this stage, after the ulcer had progressed during five weeks of active treatment, a diagnosis of Mooren's ulcer was made. Besides the corneal opacity, which was healing at the periphery but was spreading slowly toward the center of the cornea and at both ends, an area of marked hypertrophy and congestion of the bulbar conjunctiva, such as H. Gifford and others have described, was present. On Jan. 29, 1931, keratotomy was performed, the cut being 4 mm. long and just in advance of the central border (fig. 2). Marked improvement in the pain and

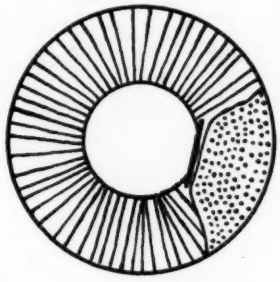

Fig. 3 (case 2).—Location of keratotomy.

Fig. 4 (case 2).—Right eye after healing.

congestion began at once. The wound was reopened on January 30 and 31 and February 2, 4, 5, 6 and 9. There was neither infiltration of the border nor staining areas. One small staining area appeared on February 13, which was touched with trichloracetic acid, following which the remaining congestion disappeared; the eye was quiet until May 17, when one small infiltrate appeared which disappeared promptly without treatment. On June 6, the eye was quiet, with vision of 20/50.

CASE 2.—Mr. L.,[12] aged 65, had an ulcer at the nasal border of the right cornea of a week's standing. It arose from the arcus senilis and extended 4 mm. toward the center of the cornea. In spite of local applications of trichloracetic acid and other local treatment, it progressed centrally and over a larger area.

12. This case was reported previously.[6]

On the sixth day of observation, the actual cautery was used by Dr. H. Gifford, who made a diagnosis of Mooren's ulcer. In spite of this treatment, progress continued, and on the thirteenth day I performed a delimiting keratotomy just central to the advancing border. The incision was kept open twice daily for ten days, iodine being applied before this each time. The ulcer healed from the periphery and never advanced beyond the incision. Corneal astigmatism was 7 diopters at first, but after two months this disappeared and vision was 20/30 without correction (figs. 3 and 4).

CASE 3.—Mrs. M., aged 30, who was apparently in excellent health, was seen in 1926. She had noted visual disturbance and pain in the right eye for about four weeks. The outer half of the cornea was opaque, with vessels extending into its peripheral portion and an infiltrated margin extending almost vertically across the cornea. There was not so much hypertrophy around the limbus as in the two preceding cases. Cauterization of the advancing margin with trichloracetic acid and phototherapy at first seemed to stop the progress of the ulcer, but after a week it began to progress farther across the cornea. A conjunctival flap was

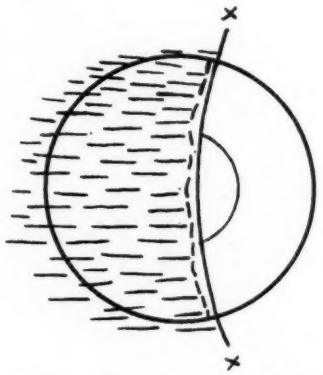

Fig. 5 (case 3).—Location of conjunctival flap.

drawn beyond the advancing border after the corneal surface was touched thoroughly with trichloracetic acid. The ulcer never progressed after this was done. Because of the application of trichloracetic acid, the flap healed in place and was allowed to remain there. After three months the eye was quiet, and vision with astigmatic correction was 20/40. While the presence of the flap gave an unpleasing cosmetic effect, it was thought safest not to disturb it, though this may be done later (fig. 5).

CONCLUSIONS

1. Delimiting keratotomy in two cases of typical Mooren's ulcer and the use of a conjunctival flap in one case resulted in arrest and healing of the ulcer, with vision of 20/50, 20/30 and 20/40.

2. I have not attempted either procedure in other cases, as no others have been seen in the active stage in recent years. In one case of H. Gifford, however, I have seen the use of a conjunctival flap fail to arrest the ulcer.

3. Delimiting keratotomy is a safe procedure and is simpler and less disfiguring than the use of a flap.

4. If a flap is employed, it should be made to cover the whole cornea, as described by Kreiker, or should extend well past the advancing border and be made to adhere to the ulcer by a thorough application of trichloracetic acid, as was done in case 3.

5. Other methods of producing hypotony, such as cautery puncture through the ulcer, may yield equally good results, but the hypotony must be maintained for at least eight or ten days and in some cases longer.

6. The effect of delimiting keratotomy seems to be due to the increased nutrition and supply of antibodies resulting from a reduction of tension and perhaps also to the epithelial barrier formed at the site of incision. Such an effect may be conceived to be of value whether the disease is infectious or degenerative.

LEUKEMIC RETINITIS

LEO J. GOLDBACH, M.D.
BALTIMORE

The purpose of this paper is to report three cases of leukemic retinitis which have recently come under my observation and the incidence of ocular complications in patients with various types of leukemia whose histories appear in the files of the Johns Hopkins Hospital.

REPORT OF CASES

CASE 1.—A white woman, aged 28, was admitted to the Mount Sinai Hospital on April 7, 1929. The family history and the past history were not significant. The disease had begun four months previously with a cold, cough, hemoptysis and fever. On admission, examination showed an area of dulness over the right side of the chest with tubular breathing, hyperactive reflexes and purpuric spots on the right breast. Roentgen examination showed pneumonic consolidation of the right lung. Blood cultures were negative. The patient recovered from these acute symptoms and was discharged from the hospital. She was readmitted three weeks later after noticing painless lumps in various parts of the body. At this time enlargement of the spleen was found; the thyroid was also enlarged, and the basal metabolic rate was + 32. On May 4, definite general glandular enlargement was noted. The tonsils were also enlarged and purple; the liver and spleen were greatly enlarged. On roentgen examination, masses were seen in the upper mediastinum. Purpuric spots appeared in the skin. The blood picture showed a white cell count varying from 22,000 to 424,000, which figure was reached shortly before death. Eighty per cent of these cells were small lymphocytes. The red cell count on admission was 3,200,000 and fell to 1,500,000 before death. The hemoglobin varied from 43 to 34 per cent.

The general medical diagnosis was: chronic lymphoid leukemia, encephalitis, drug poisoning, secondary anemia, septic pneumonia and endocarditis.

Examination of the eyes showed pale conjunctivae with hemorrhagic areas in the lower lids. The media were clear. In the fundi there were numerous radiating, flame-shaped hemorrhages, with small, darker, irregularly round hemorrhages, some of which showed whitish centers. Numerous fresh hemorrhages appeared in the fundi during the patient's hospitalization, and there were frequent extravasations of the blood in the skin of the upper and lower lids.

The patient died on June 10, 1929.

CASE 2.—A boy, aged 12 years, was admitted to the hospital on Feb. 11, 1930. At the age of 50, the mother had heart trouble. At the age of 69, the father had hypertension. The patient's general health had always been good. He had acute rheumatic fever in January, 1929.

For the past few months the patient had been troubled with palpitation and shortness of breath on exertion. There had been slight swelling of the ankles

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From the Wilmer Ophthalmological Institute, Johns Hopkins Hospital.

Read before the Ophthalmological Section of the Baltimore City Medical Society, February, 1933.

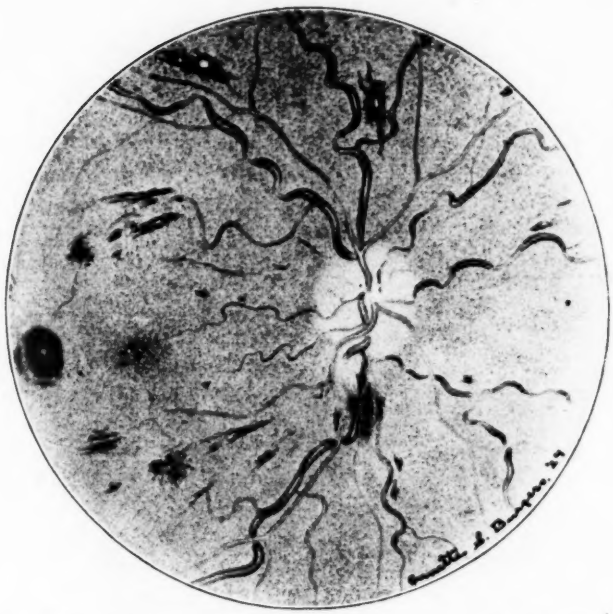

Fig. 1 (case 1).—Leukemic retinitis showing numerous radiating flame-shaped hemorrhages.

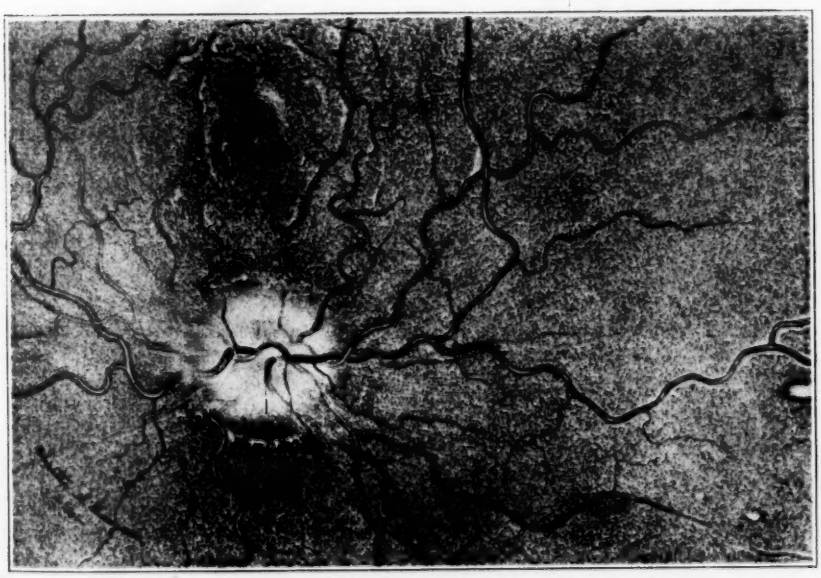

Fig. 2 (case 2).—Leukemic retinitis showing elevation of the disk 3 diopters, with whitish plaques surrounded by a reddish halo.

when standing. At Christmas time the abdomen began to swell, and remained enlarged. At the time of admission he noticed a mass in the abdomen. He began to have pain in his left eye about the early part of February. Three days prior to admission he began to have attacks of dizziness, with nausea and vomiting. Palpitation became severe.

Examination showed an enormously enlarged spleen, subcutaneous nodules (rheumatic or leukemic ?) over the tibiae and undernutrition. The white blood cells numbered 500,000; the differential count showed 64 per cent myelocytes. Roentgen examination of the chest showed the lungs to be clear; the head was normal.

The medical diagnosis was chronic myeloid leukemia.

Examination of the eyes showed them to be normal externally. Ophthalmoscopically, there was marked elevation of the disk of the right eye, the apex being brought into view with a + 3 sphere. There was enormous tortuosity of the vessels, with some retinal edema. The veins and arteries were practically the same color. The disk was quite red and edematous, and the cup was practically filled. Around the entire periphery of the fundus the vessels showed a perivascular infiltration, giving the picture of white stripes along the vessels. In the periphery of the retina there were also whitish plaques, many of which were surrounded by a reddish halo. Practically the same condition existed in the left eye. Vision in the right eye was 20/30; with glasses, 20/30 + 4. Vision in the left eye was 20/30—2; with glasses, 20/30 + 4. The visual fields were normal.

The patient was given radium therapy, and on discharge the condition was much improved, and the spleen had decreased in size. The white blood cells fell to 15,000, and the myeloid forms decreased from 64 to 1 per cent. There was a concurrent rise in the hemoglobin from 43 to 62 per cent. The patient was discharged on March 20.

On that date the eyegrounds had improved markedly, and the disks were only slightly blurred.

Case 3.—A colored boy, aged 10 years, was admitted to the Johns Hopkins Hospital on Feb. 21, 1931. The family history was unimportant except that the father and mother were receiving antisyphilitic treatment. The past history was irrelevant, the child having had no illnesses of any kind prior to the present illness. This illness began in November, 1930, the chief symptoms being pain in the knees, chest and head, frequent transient periods of elevation in temperature and frequent attacks of epistaxis. During the month prior to admission he had gradually grown weaker and suffered from shortness of breath. The parents stated that at each attack of epistaxis the blood was lighter in color and more watery than before. On admission examination showed an anemic, dyspneic child who was apparently very ill. The membranes of the mouth and throat were pale; the cervical, axillary and inguinal glands were palpable. There was an area between the sixth and seventh intercostal spaces on the right side, in the anterior axillary line, which was extremely sensitive to pressure. There was a soft systolic murmur at the apex; the liver and spleen were palpable. The hemoglobin was 20 per cent; the red blood cells numbered 750,000; the white blood cells, 26,800; the polymorphonuclears were 5 per cent, and the lymphocytes, 94 per cent; the platelets were diminished. The clinical diagnosis was lymphatic leukemia.

Examination of the right eye showed that the bulbar excursions were good. The conjunctiva was pale. The cornea, media and lens were clear. The pattern of the iris was normal. There was no migration of pigment cells. The outline of the disk was blurred, and the disk was elevated 2 diopters. There was a subhyaloid hemorrhage on the nasal side of the nerve, and another one about twice

its size above and temporally, covering the retinal vein. There were a few striate hemorrhages between the vessels. The arteries in the periphery were lessened in caliber, and as they neared the nerve head they had a beaded appearance; here and there they were lost, owing to the edema of the retina. The veins were engorged and tortuous, and as they neared the nerve head there was a distinct bending, with a white streak along the wall of the vessels. The edema around the nerve head obscured the outline of the vessels.

Examination of the left eye showed clear media. The outlines of the nerve were indistinct, more so toward the nasal side. The disk was elevated about 1 diopter. There was a subhyaloid hemorrhage in the upper portion of the fundus. The edema of the retina was more marked than in the right eye. The vessels were not clearly defined, and the veins were more tortuous. There were

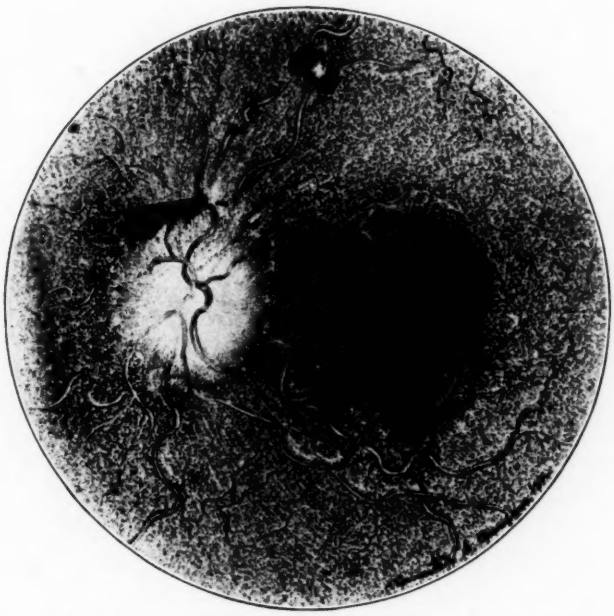

Fig. 3 (case 3).—Leukemic retinitis. Elevation of the disk and subhyaloid hemorrhage with engorged and tortuous veins.

white streaks along the vessels and white areas throughout the fundus. The left fundus did not show as many subhyaloid and striate hemorrhages as the right, but the outlines of the nerve were more indistinct, apparently owing to the increased edema of the retina.

The impression was that the patient had papilledema of the nerve heads, with subhyaloid and striate hemorrhages, elevation of the disks of 2 diopters, white streaks along the walls of the vessels, increased caliber of the veins, decreased caliber of the arteries, tortuosity, no apparent disturbance of the choroid, no change in the choroidal pigment and veil-like opacity of the retina.

The patient's course in the hospital was steadily downhill in spite of transfusions and intravenous injections of adenine sulphate. He died on May 8, 1931. During his stay in the hospital, however, the hemorrhages noted in the fundus cleared up so that only vestiges remained.

TABLE 1.—*Analysis of Cases from the Johns Hopkins Hospital*

	Acute Lymphatic Leukemia	Chronic Lymphatic Leukemia	Acute Myeloid Leukemia	Chronic Myeloid Leukemia
Retinitis:				
Hemorrhages	8	2	4	17
Hemorrhages and exudates	1	..	..	3
Retinal and subhyaloid hemorrhages and white patches	1	..	..	..
Exudates	..	..	1	..
Hemorrhages and white patches	..	..	..	1
Pallor	..	..	..	3
Pigmentation	..	..	..	1
Retinal vessels:				
Tortuosity	4	9	4	25
Changes in caliber	..	..	..	1
Pallor	..	1	..	..
Nerve head:				
Optic neuritis	9	3	1	17
Pallor	2	3	..	2
Choked disk	..	..	..	1
Hyperemia	..	..	..	1
Edema	..	..	..	1
Pupils:				
Sluggish reaction to light and anisocoria	2	..	..	..
Sluggish reaction to light	1	..	..	2
Anisocoria	1	..	1	2
Pupillary reaction to light retarded	..	4	..	1
Mydriasis	..	..	1	2
Convergence retarded	..	..	..	1
No pupillary reaction to accommodation	..	..	..	1
Pupillary reaction retarded—light and accommodation	..	..	1	..
Conjunctivitis	9	6	2	28
Scleritis	7	6	1	19
Blepharitis	1	3	2	6
Glaucoma	1	..	..	..
Iritis	..	3	..	..
Keratitis	..	4	..	1
Cataract	..	2	..	1
Paralyzed internal rectus muscle and nystagmus	..	1	..	..
Exophthalmos	..	..	..	3
Transitory external strabismus	..	3	..	4

TABLE 2.—*Analysis of Cases from the Harriet Lane Hospital*

	Acute Lymphatic Leukemia	Chronic Lymphatic Leukemia	Acute Myeloid Leukemia	Chronic Myeloid Leukemia
Retinitis:				
Hemorrhages	3	1	..	..
Hemorrhages and leukemic infiltration	1	..	..	..
Pallor	1	..	1	..
Retinal and subhyaloid hemorrhages	1	..	..	..
Exudates and hemorrhages	..	..	..	..
Edema	..	..	..	1
Retinal vessels:				
Tortuosity	3	..	..	..
Nerve head:				
Optic neuritis	6	1	..	1
Pallor	2	..	..	1
Pupils:				
Exophthalmos	..	1	..	..
Conjunctivitis	16	1	4	5
Scleritis	2	..	1	1
Blepharitis	7	..	1	1
Nystagmus	..	..	..	1

An analysis of the various types of leukemia seen in the Johns Hopkins Hospital revealed 45 cases of acute lymphatic leukemia, of which 25, or 55 per cent, gave positive ocular findings. There were 42 cases of chronic lymphatic leukemia, of which 28, or 62 per cent, showed positive ocular findings. There were 9 cases of acute myeloid leukemia, of which 8, or approximately 90 per cent, showed positive ocular findings, and 143 cases of chronic myeloid leukemia, of which 82, or 60 per cent, showed positive ocular findings.

Table 1 shows the exact distribution of the various ocular lesions in the various types of leukemia.

In the Harriet Lane Home, the Children's Department of the Johns Hopkins Hospital, there were 53 cases of the various types of leukemia. Table 2 shows the occurrence and distribution of ocular complications in this group of patients.

COMMENT

Acute leukemia, while comparatively rare, is the prevailing type of leukemia in children. Although no organism has been demonstrated as the causative agent in this type of leukemia, nevertheless the disease bears many characteristics of an acute infectious process. Two forms of acute leukemia are recognized—the myeloid and the lymphoid. The primary change in both types probably occurs in the bone marrow, and the lymphatic structures are secondarily involved. In the lymphoid type the predominating cells in the bone marrow are immature lymphoid cells or lymphoblasts. In the myeloid type the predominating cells in the bone marrow are immature myeloid cells or myeloblasts. The onset is usually abrupt, with ulcerative pseudomembranous involvement of the mouth, gums and tonsils. Local hemorrhages, especially those in the kidneys, are frequently observed early in the disease. The fully developed clinical picture strikingly resembles an acute infection with pallor, loss of weight, irregular fever and hemorrhages from the mucous membranes, skin, retina and viscera. Enlargement of the lymph nodes, tonsils and spleen is usually moderate but is commonly observed. The leukocyte count is usually high, from 50,000 to 200,000. In the lymphoid type of the disease the predominating cells are either small lymphocytes or large mononuclear cells with a greater amount of cytoplasm, but less deeply stained, and sometimes a slightly globulated nucleus, an immature lymphocyte or a lymphoblast. In the myeloid form of the disease the predominating cells are nongranular myeloblasts, although other cells are frequently found. The more acute the disease the greater is the predominance of myeloblasts with myelocytes. There is a rapid decrease in the number of red cells, and the red count frequently falls to

1,000,000 or less. The number of platelets is also low. The disease usually terminates fatally within a few months.

Chronic lymphoid leukemia is a disease of insidious onset and is usually manifested first by an enlargement of the lymph nodes and a gradual loss of weight and strength. There is usually a slight increase in the size of the liver and the spleen, although much less than is usually observed in myeloid leukemia. Febrile attacks occur from time to time. The disease is characterized by leukemic infiltrations in various parts of the body. These infiltrations may occur in the lungs, the ear, the eye, the orbit and the skin. The blood picture is quite characteristic. The leukocyte count is greatly increased, but less than in myeloid leukemia, the count not often exceeding 100,000, and sometimes being as low as 30,000. The increase consists exclusively of lymphocytes, chiefly small

TABLE 3.—*The Blood Picture*

	Red Blood Cells		White Blood Cells		Hemoglobin, per Cent		Monocytes, per Cent		Myeloblasts, per Cent	
Type of Leukemia	High	Low	High	Low	High	Low	High	Low	High	Low
Acute lymphatic	6,500,000	780,000	832,000	4,000	87	12	98	8	..	..
Chronic lymphatic	5,800,000	700,000	471,000	2,800	87	35	95	23	..	..
Acute myeloid	3,400,000	1,000,000	492,000	4,480	70	14	95	3.4	99	2
Chronic myeloid	7,720,000	1,200,000	930,000	1,720	105	6	71	3	98	2

Distribution	Per Cent	Number
White patients	89.6	
Colored patients	10.4	
Male patients	64.1	
Female patients	35.9	
Died	63	
Improved	12.1	
Treatment with roentgen rays		38
Treatment with radium		54

ones. There is usually a marked decrease in the red blood cells, the red count frequently falling to 2,000,000 or less, many nucleated forms appearing in the picture. The disease is practically always fatal, but may continue for three or four years before death takes place. Its course is frequently marked by remissions and exacerbations.

Chronic myeloid leukemia is a disease of insidious onset, the first change usually noticed being a gradual abdominal enlargement with pain in the side, increasing weakness and hemorrhage. Characteristic of the disease is the extreme enlargement of the spleen. The disease is also characterized by gradual weakness, slowly developing anemia, hemorrhages from various parts of the body, especially from the nose, and various symptoms referable to the digestive tract. Hemorrhages and leukemic infiltrations occur in the eye and the internal ear, and also in other portions of the body. The changes in the blood are of first importance and are distinctive. The white blood count is usually

increased from 100,000 to 500,000 cells per cubic millimeter, and at times to an even greater figure. All varieties of white cells are found, but especially there is a great number of myelocytes, which may constitute from 5 to 50 per cent of the total leukocytes. Red cells are usually decreased to 2,000,000 per cubic millimeter or less, but these changes generally occur late in the disease. The color index is low in most cases. The disease is usually incurable, but remissions are not uncommon. Temporary improvement frequently occurs under treatment.

In all the cases of leukemia the ages of the patients varied from 21 months to 74 years, the mean average being 43 years.

Leukemic retinitis was apparently first noted as a clinical entity by Liebreich in 1861. The changes in the retina appear to bear no relationship to the number of white cells in the circulating blood. In some cases with a greatly increased cell count the retina may appear normal, while in other cases with a low blood count a destructive chorioretinitis may be present.

Leuterich described the ophthalmoscopic picture as follows:

> The retinal and choroidal vessels show pallor, especially the retinal veins, associated with increased calibre, bending, beading, and hemorrhages; secondly, haziness of outlines of the disc margins with irregular white spots close to the macular area; thirdly, exudates resembling those seen in the renal retinitis, but differing in their locations.

The pathologic changes in the retinal lesions have been studied by numerous observers, especially by Deutschman, Nagoya and Verderame.[1] Deutschman showed that hemorrhages occurred in the nerve fibers, in the ganglion cell layer of the retina, with infiltration into the external sheath of the optic nerve. Nagoya described the choroid as being excessively infiltrated and thickened to four times its normal size. This increase was more pronounced centrally, gradually decreasing toward the periphery. The infiltration was caused by a prodigious number of round cells. All of the tissue substances were so crowded with these new cells that only a suggestion of the real structure of the choroid could be recognized. These cells were usually myelocytes, lymphocytes, isolated eosinophils, granular cell forms and occasional nucleated red cells. Verderame [1] described the pathologic changes in a leukemic eye as follows:

> The choroid has a normal appearance as far as the equatorial region. From this point a slightly irregular widening can be recognized, due to a leukocytic increase in the stroma, caused chiefly by the accumulations of white cells. These circumscribed areas are in parts rather long, although some of them are more

1. Verderame, P.: Ueber Augenveränderungen bei der akuten und chronischen Leukämie, Virchows Arch. f. path. Anat. **200**:367, 1910.

round. On close examination of these areas, distended veins filled with white cells are seen, between which are many red corpuscles. Other vessels may contain white blood corpuscles exclusively. In other areas, especially the outer layers of the choroid, considerable numbers of red blood cells can be recognized with the white blood cells. In the transverse section of the arteries the white cells appear to lie in the stroma and form close cell-tunics around the lumen. In other places where the accumulation of cells is more extensive, little of the vessel wall can be recognized. When stained by Weigert's method it can be seen that this is an intravascular cell mass formation. The retina as a whole is thickened and in certain areas has the appearance of irregular elevations; the edema is not uniform. There is a diffuse sclerosis of the nerve fibers. The protuberant elevations in the inner layers of the retina are round and spindle-shaped, the smaller of these taking in only the layers of the nerve fibers and the ganglion cells. The retinal arteries are not distended nor abnormally filled; they contain red blood cells for the most part, although around many of them there are large accumulations of white cells. The supporting tissue of the retina is slightly hypertrophic; the Müller fibers are thickened. The optic papilla is edematous and there is cellular infiltration of the lamina cribrosa. The central vessels are not abnormally filled and their walls not thickened.

The hemorrhages in various parts of the body and in the eye are characteristic of all forms of leukemia, and the hemorrhages in the fundus do not appear to be especially characteristic in any of the various types of leukemia. Hemorrhages occurring in diseases of the hematopoietic system are probably due to defective nutrition of the capillary endothelium secondary to poor oxygenation and the changes in the chemical composition of the blood.

The peculiar color of the fundus noted in many cases of leukemic retinitis is not characteristic. When present, this color varies from yellow to light pink. It appears to be dependent on the type and location of the hemorrhages, the degree of infiltration of the choroid and the condition of the retinal circulation. An infiltration of the choroidal retinal vessels together with a retarded circulation apparently hastens the accumulation of white cells in the retina, for the leukocytes appear to have a great tendency to adhere to the walls of the vessels, especially in the capillaries. Oeller took the point of view that this can occur only at the expense of the lymph channels.

The changes in the caliber and tortuosity of the veins are significant of the altered blood picture. The blood flow is retarded chiefly in the veins and capillaries, with a resultant seepage of the unformed elements of the blood from the capillaries and secondary pressure on the perivascular lymph sheaths; this produces secondary edema of the retina, which may be further aggravated by the location and character of the hemorrhages. The obstruction of the lymphatics, the retarded circulation, the dilated and tortuous veins, the decreased caliber of the arteries, the extravasation of blood plasma and the relatively unyielding lamina cribrosa tend toward local edema with congestion of the disk.

Nagoya stated that the important question is how this congestion of the optic nerve takes place in the absence of all symptoms of brain pressure. It appears that the papilledema observed in leukemic retinitis must be regarded as a local phenomenon due to the factors mentioned, and accelerated in those instances in which hemorrhage takes place in the sheaths of the optic nerve.

The differential diagnosis is not difficult in most cases. In leukemic retinitis the hemorrhages usually show a small white spot in the center, probably a local collection of white cells. In diabetic retinitis the exudates do not assume a stellate appearance, but are rather round and sharply defined and are lustrous. The hemorrhages usually occur in the fiber layer of the retina. In septic retinitis the picture may be similar to that observed in leukemic retinitis. The extravasations are round or oval, are situated close to the papilla and have little relationship to the large vessels. Hemorrhages are frequently seen. Bacterial emboli with circumscribed inflammatory reactions are sometimes found. In progressive, pernicious anemia hemorrhages are more frequent and are usually large and lack the hemorrhagic halo commonly seen in leukemic retinitis.

Verderame has demonstrated glycogen in several of his cases of leukemic retinitis. The glycogen was demonstrated in the vessels as well as in the retina. In the blood it occurred chiefly in the leukocytes, sometimes in the nuclei and sometimes in the margins of the cells. Small deposits of glycogen were also found in other white cells and just outside the cells. In the retina the glycogen was distributed chiefly in the nerve fiber layer. The glycogen reaction was also distinct in the walls of the retinal vessels. While recent investigations have demonstrated that glycogen may appear in the blood in normal conditions, nevertheless the demonstration of glycogen in the choroid in leukemia as well as in acute inflammatory processes is of interest and strengthens the assumption that there is a definite toxic factor in leukemia.

SUMMARY

Three cases of leukemia are reported: 1 of chronic lymphatic leukemia, 1 of chronic myeloid leukemia and 1 of acute lymphatic leukemia. These 3 cases were complicated by concomitant retinitis. A statistical study of the ocular complications in the various forms of leukemia which have occurred in the Johns Hopkins Hospital is presented. The factors producing the changes in the retina in leukemia are discussed. Infections were noted 26 times in acute lymphatic leukemia, 23 times in chronic lymphatic leukemia, 2 times in acute myeloid leukemia and 38 times in chronic myeloid leukemia.

Clinical Notes

A NEW MOTION PICTURE CAMERA FOR RECORDING OPERATIONS ON THE EYE

H. F. Pierce, Ph.D., Baltimore

This equipment was designed to afford an adequate and convenient means of recording the technic of surgical operations, while requiring the minimum of training and effort on the part of the photographer. The apparatus and methods have been so simplified that an hour's instruction in focusing and adjusting the camera should enable one to make acceptable pictures of the operative procedures with which one is familiar. Such films are valuable as records, they are an aid to the surgeon in studying and modifying his technic, and they are useful in teaching.

The camera uses 16 mm. film in 100 or 200 foot (30.5 or 61 meter) rolls, permitting four or eight minutes of recording at the standard speed of sixteen frames per second. The mechanism is driven by an electric motor having a dynamically balanced armature, the gyroscopic action of which tends to counteract the vibration of other and lighter moving parts. The motor is controlled by a friction governor affording speeds of from eight to forty-eight frames per second, which permits the making of slow-motion pictures. This motor has manifest advantages over the spring-driven types, since it obviates the usual rewinding of the spring several times during an operation. A footage indicator on the camera housing shows the amount of film used and the amount still available. The camera is commendably silent in action.

Four lenses in spiral focusing-mounts are fitted to a revolving turret on the front of the camera. They have focal lengths of 15, 25, 75 and 100 mm., and with the camera 75 cm. from the field of operation they cover fields of 50, 30, 9 and 6 cm. respectively. Thus, without changing the distance between the camera and the subject, pictures of larger or smaller field and of lesser or greater magnification can be made, and by revolving the turret, one may switch instantly from a broad view of the operative field to a "close-up" showing only the eye and the structures immediately adjacent to it.

The 75 and 100 mm. lenses are focused through side tubes, each carrying an eyepiece, a ground glass screen and a right-angle prism. The prism is slid into the optical axis of the camera for focusing and withdrawn when the picture is to be taken. The 15 and 25 mm. lenses are focused through a similar device located on the side of the camera, and by rotation of the turret they are swung into the optical axis of the camera for use. The parallactic error (3 cm.) is easily allowed for in focusing these two lenses, since they have large fields and considerable depth of focus.

From the Wilmer Institute of the Johns Hopkins University and Hospital.

Figure 1 shows the camera mounted on a standard tripod for general use, while figure 2 illustrates the camera, camera suspension and lighting equipment as they appear in the operating room. The camera suspension and lighting equipment form an integral part of this simplified system for making motion pictures.

The vertical standard of the operating lamp carries a figure-4 arm and camera mount, attached by a ball bearing ring at the top and a bronze sleeve bearing at the bottom of the standard. The camera may be

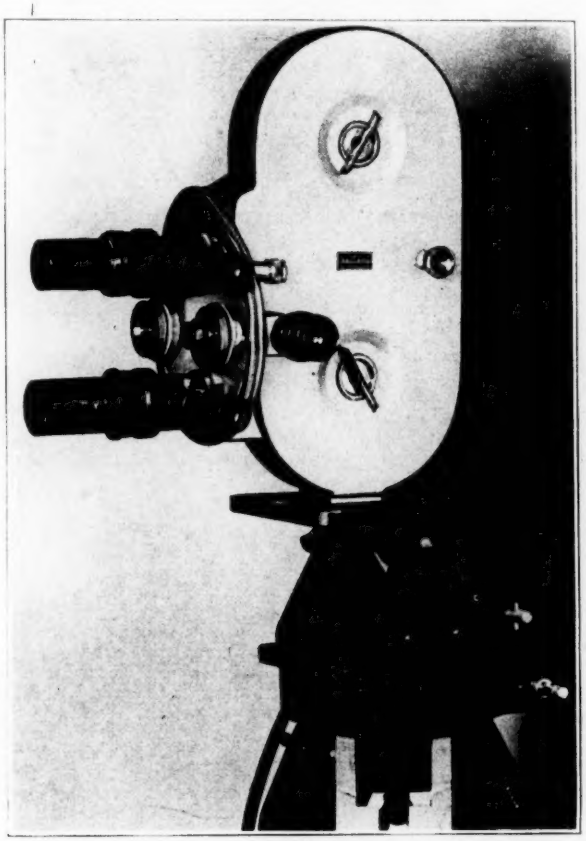

Fig. 1.—The camera mounted on a standard tripod for general use.

moved along the horizontal arm, rotated vertically and horizontally in its mount and clamped rigidly in any position. The arm may be swung completely around the standard of the operating lamp, allowing pictures to be taken from any angle.

The operating lamp has been modified by eliminating all adjustments except the raising and lowering of the light-arm and the rotation of the light-arm about its vertical axis. The two lights are arranged to project their beams downward and inward at angles of 30 degrees from the vertical and 60 degrees from each other. They are focused on the operative field simply by raising or lowering the light-arm, once the patient's eye has been centered under the vertical axis of the lamp standard.

Under the middle of the light-arm is mounted a smali spot-light which projects a narrow beam downward in line with the vertical axis of the lamp standard. This affords an easy and satisfactory means of centering the eye in the axis of rotation of both the light-arm and the camera-arm, and facilitates focusing the camera and lights on the operative field.

The method of using the equipment is simple. The patient, prepared on the operating table in an adjoining room, is wheeled into the operating room, and his eye is centered under the spot-light. The spot-light is then turned off, the table is adjusted to the height of the surgeon, the

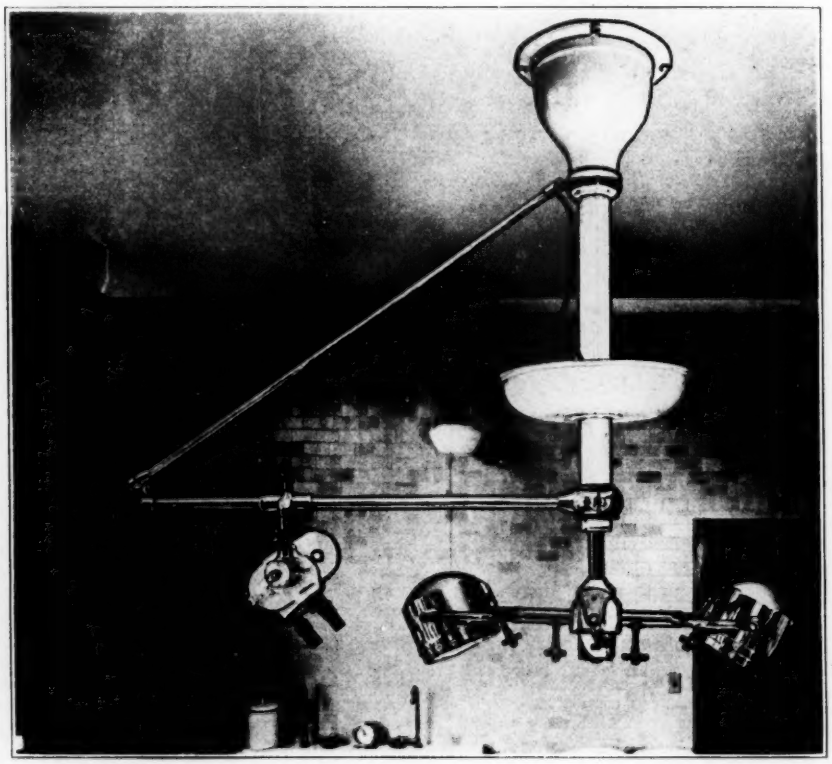

Fig. 2.—The camera, camera suspension and lighting equipment as they appear in the operating room.

operating lights are turned on, and the light-arm is raised or lowered until both beams are focused on the field. The photographer stands on a stool, focuses and locks his camera and makes long or short runs as required by the technic of the operation. Should it be necessary to photograph the action from a different angle the camera-arm is swung about its vertical axis to the new position. Since both the camera and the lights rotate about a vertical axis passing through the patient's eye, they are always trained on the operative field regardless of the angle from which the picture is taken.

The choice of film for this work depends somewhat on the subject. In general, supersensitive panchromatic film is most suitable, since it shows good detail in the reds, while its high speed under tungsten fila-

ment lamps allows the use of comparatively little light. The usual operating lamps provide more than enough illumination, and a good hand-operating lamp gives satisfactory results, even with lenses working at apertures of f. 4.5 or smaller. High speed lenses are neither essential nor advantageous, since those working at apertures greater than f. 3.5 have neither the depth of focus nor the flatness of field desired in this work.

Careful editing and judicious tilting are necessary for the production of good films, and once the film is complete and approved it is advisable to have a duplicate made from it. Duplicates, having no cemented joints due to editing, run smoothly in the projector, and when they are badly worn they may be replaced at moderate cost by printing from the original. If stored in proper humidors the originals will remain in good condition indefinitely.

With the equipment here described it is unnecessary to "stage" an operation for the camera. No special lighting is required, and no valuable floorspace is taken up by a cumbersome tripod or camera stand. The remoteness of the camera from the field and its ease of manipulation permit the photographer to record the operation with a minimum of interference with the work of the operating team. Therefore the film shows the operation as it is usually performed, and not as it is sometimes "staged" to meet the requirements of the photographer.

FAMILIAL BILATERAL ANKYLOBLEPHARON

Report of a Case

A. FINE, M.D., BROOKLYN

Because of the extreme rarity of the congenital anomaly familial bilateral ankyloblepharon and the paucity of references to it in American and in foreign literature, I feel that a description of the following case is worthy of written record. The condition present is a familial, bilateral ankyloblepharon with aplasia of the lacrimal passages and structures of the inner canthus.

Ankyloblepharon [1] is a condition in which there exists a fusion of the margins of the lid, either in part or in toto, and is either congenital or acquired. In the congenital variety, this union of the margins of the lids is epithelial, and there is no symblepharon. In the acquired type, resulting from burns, trauma or serious infections, like diphtheria, the body of the lids is fused, and almost always there is an associated symblepharon. The term "cryptophthalmus" has been confused in the literature with ankyloblepharon. In the former, the lid has no margin; a fold of skin passes from the forehead to the cheek without differentiation into lids.

Presented before the New York Academy of Medicine, Section of Ophthalmology, Jan. 18, 1932.

1. Wood, C. A.: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 4, p. 2793.

REPORT OF A CASE

A boy, aged 5 years, Italian, was brought to me by his mother for the treatment of strabismus and a persistent tearing of both eyes, both conditions dating back to infancy. On gross examination, I noted the dull expressionless face, the pseudostrabismus (a true squint was ruled out by the usual muscle tests), the absence of a normal inner canthus, the pinched nares, the maldevelopment of the nasal bridge and what may have been a characteristic feature, the fissured lips.

Close study revealed a web of skin joining the canaliculi in such fashion as to hide the possible presence of a caruncle or plica semilunaris. The punctae were properly located, and the canaliculi were patent, but the remainder of the lacrimal passage was obstructed. The internal canthal ligaments were strongly developed, and a faint horizontal line was to be seen marking the site of the fused lids medially. There were no other congenital anomalies, and the eyes were otherwise normal. The Wassermann reaction was negative, and there were no signs of congenital syphilis. A similar condition, but less pronounced, was seen in the patient's younger brother, and a suggestion of a narrowing of the inner canthus appeared in the mother.

Fig. 1.—Original condition prior to operation.

According to Ida C. Mann,[2] the lids are normally fused in the fetus. The fusion is purely of epithelial character, and exists from about the tenth week to the seventh month of intra-uterine life, when the lids gradually undergo separation. It is held that this separation is brought about by the growth of the eyeball exerting pressure on the fused lids, and that when such a failure of separation occurs, the eye is usually small and undeveloped. This is not borne out in my case, as there was no suggestion of microphthalmia.

A diagnosis of congenital ankyloblepharon was warranted by the symmetrical appearance of the lesion, together with the weblike nature of the fusion, the absence of scars and other evidence of trauma or inflammation and, of course, the history of its origin and familial association. From epicanthus, it was differentiated by the appearance, in the former, of a semilunar fold of skin, overlying a normally developed inner canthus, which can be made to disappear by pinching up a portion of skin at the root of the nose. The pseudostrabismus, which it simulated, was unmasked after the operation.

At the Herman Knapp Memorial Eye Hospital, I did a canthoplasty to correct the blepharophimosis. A horizontal incision, about 7 or 8 mm. long, was made along the line of fusion. The conjunctiva was dissected up, so that the angle of the conjunctional wound was sutured to the angle of the cutaneous wound, and

2. Mann, Ida C.: The Development of the Human Eye, New York, The Macmillan Company, 1928, p. 259.

then the conjunctiva and skin were united by interrupted sutures at the margin of the lids. This opened up the internal canthus and revealed a rudimentary caruncle and plica semilunaris. To obtain an enhanced effect, I did a secondary operation about a month later. This consisted in removing a crescentic portion of the skin on each side of the root of the nose.

Instillation of iodized poppy seed oil 40 per cent followed by roentgenography was done in an effort to visualize the contour of the lacrimal passages, but this attempt was unsuccessful because of the obstruction encountered beyond the canaliculi.

In the American literature, Oppenheimer,[3] in 1924, reported a case of medial congenital ankyloblepharon in a woman, aged 66. Her complaint was that of tearing over a period of many years. He noted the absence of a plica semilunaris and caruncle on both sides. The punctae and canaliculi were normal, but very narrowed, and syringing through to the nose was impossible. The observation is interesting that she was referred from the dermatologic department, where she was treated for "sores of the mouth."

Fig. 2.—Result of operation.

Ford,[4] at the Royal Society of Medicine, in 1925, showed a father and daughter with congenital ankyloblepharon. The father had an absence of the levator palpebrae superioris, and the child had had the external canthus slit and was able to open the eyes much better.

Van der Hoeve,[5] in 1929, described a case of "congenital elongation of the inferior-lacrimal canaliculus, with internal ankyloblepharon." Neither his description nor the picture of his patient conforms to my notion of an ankyloblepharon.

Other cases appear in the literature, described by Begle,[6] Cobbledick[7] and McMullen,[8] dealing with congenital malformations of the canthi, but the descriptions are not of ankyloblepharon.

3. Oppenheimer, E. H.: Am. J. Ophth. **7**:788 (Oct.) 1924.

4. Ford, R.: Proc. Roy. Soc. Med. (Sect. Ophth.) **18**:25, 1925.

5. van der Hoeve: Bull. et mém. Soc. franç. d'opht., 1929, p. 210.

6. Begle, H. L.: Bildungsanomalie der innerin Lidkante, Ztschr. f. Augenh. **31**:235, 1914.

7. Cobbledick, A. S.: Congenital Bilateral Deformity of Inner Canthus, Proc. Roy. Soc. Med. (Sect. Ophth.) **6**:62, 1912-1913.

8. McMullen, W. H.: Congenital Malformation of Outer Canthus, Proc. Roy. Soc. Med. (Sect. Ophth.) **13**:85, 1919-1920.

SUMMARY

A rare interesting congenital anomaly of the lids, but little mentioned in the literature, is described, and the references on the subject are brought up to date.

DIPTEROUS LARVA PASSING FROM THE OPTIC NERVE INTO THE VITREOUS CHAMBER

MICHAEL PRICE DEBOE, M.D., MIAMI, FLORIDA

On May 29, 1933, E. M. Kroger, a white man, aged 48, came to the office because of a sudden attack of blindness in the right eye, which had occurred one hour before.

The personal history and the family history were irrelevant, except that he had lived in the tropics for a number of years. He had never had any tropical diseases or any other sickness. There was no history of injury to the eye or of being struck in the eye by a fly. There had been no break in the skin or mucous membrane recently, except for a tick bite on the body two weeks previously.

Examination of the eye showed a partially dilated pupil which was not responsive to direct light, but retained its power of consensual reaction.

Ophthalmoscopic examination revealed a small black point in the center of the optic disk moving actively in a circle 0.5 mm. in diameter. This black point broke through the retina and revealed itself as the head of a dipterous larva. The larva crawled out of the nerve head into the vitreous chamber. The time from the first observation to its exit into the vitreous was not longer than twenty seconds.

A hemorrhage from the nerve head followed immediately, and the larva was completely covered. The patient could then see well enough to count fingers at 1 meter. It was but a few hours, however, before the eye was blind again as a result of optic neuritis.

On the second day a pseudopoid movement in the clot could be seen, but this ceased on the third day. The clot extended from the disk along the floor of the vitreous chamber almost to the lens.

By the sixth day the clot showed definite signs of absorption, but the larva was not seen again until the eleventh day, when it could be clearly seen suspended in the vitreous about 7 mm. in front of the retina and about 2 mm. above the horizontal meridian. At the time of writing it was still in the same location and position. It looked as though it were suspended in a show case. The larva was about 3 mm. long and legless, and had seven segments. It had a small black head and a pointed tail.

On the twelfth day acute cyclitis developed. The tension increased to 32 mm., and the pupil contracted. Atropine was used freely, and the pupil dilated promptly. The tension disappeared as soon as the pupil dilated.

The optic neuritis which followed the exit of the larva cleared up in the course of two weeks and left the nerve atrophic, with a total loss of vision.

The patient has been examined thoroughly. Examinations of the blood, feces and urine were made, and no ova of any kind were found.

Specimens of blood taken during the day and also at night were examined for parasites, but none was found. Physical examination, determinations of the blood pressure and examinations of the heart, lungs, abdomen and other organs, gave negative results.

Fig. 1.—Drawing illustrating the dipterous larva in the vitreous chamber.

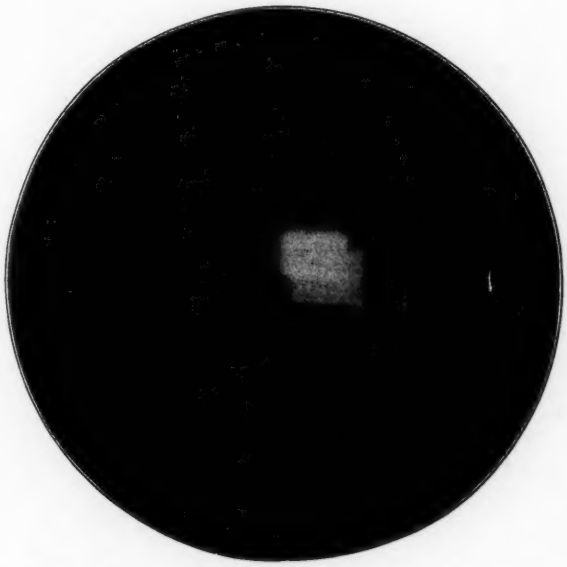

Fig. 2.—Photograph showing the dipterous larva in the vitreous chamber. It is about twelve times normal size.

Dr. B. L. Whitten and Dr. Nelson Miles Black of Miami, Fla., and Dr. Thomas Hayes Curtin of New York observed the case. Dr. C. H. Curran, of The American Museum of Natural History, New York, cooperated in establishing a more definite classification of the larva; it will be given in a subsequent report.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Scientific Exhibit, Cleveland Session.—Application blanks are now available for space in the Scientific Exhibit at the Cleveland Session of the American Medical Association, June 11 to 15, 1934. The Committee on Scientific Exhibit requires that all applicants fill out the regular application form and requests that this be done as early as convenient. The final date for filing applications is Feb. 26, 1934. Any person desiring to receive an application blank should address a request to the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago, Ill.

Lectures on Ophthalmology.—A series of comprehensive lectures on ophthalmology will begin on Jan. 1, 1934, in Chicago. Two hundred hours of didactic instruction will be given by fifty-three instructors from the various universities and hospitals; the lectures will be divided evenly between the fundamental and clinical branches of ophthalmology.

Practically all of the lectures will be given at the Illinois Eye and Ear Infirmary, and they will be open to all ophthalmologic interns and residents in the Chicago hospitals.

The second edition of the "Indicia Ophthalmologica," listing the ophthalmic surgeons of the world, has been published, and copies may be obtained from the editor, Mr. Eduard Ijdo, Hooglandsche Kerkgracht, 4 Leyden, The Netherlands. The price is 7 Swiss francs.

SOCIETY NOTES

National Society for the Prevention of Blindness.—The annual meeting of this society was held on Friday, Nov. 24, 1933, in the Russell Sage Foundation Building, New York. Mr. Preston S. Millar, president of the Electric Testing Laboratories and one of the directors of the Society, gave the principal address on the subject "Light and Vision." Following the annual meeting, on November 25, a luncheon meeting devoted to the subject of medical-social eye service was held in conjunction with the North Atlantic District of the American Association of Hospital Social Workers, the Medical Social Service Section of the Welfare Council of New York City, and the Department for Prevention of Blindness of the New York State Commission for the Blind.

Among the interesting events of the year were: the Society's celebration of its silver anniversary in May; the active campaign against syphilis as a cause of blindness; the increase in the number of sight-saving classes, with three colleges and universities offering special training in this work; and the growth in the number of workers in the medical-social eye service since the first course in 1931 (of eleven

trained workers, nine came from the Massachusetts Eye and Ear Infirmary, Boston, and two from the Washington University Clinics and Allied Hospitals, St. Louis).

Society of Ex-House Surgeons of the Manhattan Eye, Ear and Throat Hospital.—A two day reunion of the Society of Ex-House Surgeons of the Manhattan Eye, Ear and Throat Hospital was held in New York on Sept. 22 and 23, 1933. The program included clinics and demonstrations at the hospital, a luncheon at the hospital and a final dinner at the New York Physicians Club.

Ophthalmological Society of Paris.—Preceding the annual meeting of the Society in Paris on November 19, there was a public session of the League for the Prevention of Trachoma, according to the following program: (1) "Trachoma and Blindness in the Southern Part of Tunisia," Dr. Talbot; (2) "Copper Citrate in the Treatment of Trachoma," Dr. Enrique Selfa; (3) "Unilateral Trachoma; Unilateral Gonorrheal Conjunctivitis," Dr. Elena Puscariu; (4) "Some Bacteriological Properties of Chaulmoogra Oil," Dr. C. Trapesontzeva and Dr. Z. Nikolskaja; (5) "Some Facts Regarding the Struggle Against Trachoma in the Countries with a Mixed Population," Dr. L. Poleff. The following papers were read before the society: (1) "Treatment of Ocular Tuberculosis," Dr. Edward Werdenberg; (2) "Localization of Brain Tumors," statutory conference conducted by Dr. Clovis Vincent; (3) "Nature and Treatment of Pterygium," statement and discussion of the report by Dr. Merigot de Treigny and Dr. Coirre.

UNIVERSITY NEWS

Dr. Harald Gjessing, of Drammen, Norway, will give the annual lectures which are a feature of the meetings of the North of England Ophthalmological Society. The lectures, with the place and date, are as follows: "The Value of Eye Symptoms in the Diagnosis of Internal Disease," University of Manchester, Jan. 5, 1934; "The Value of the Ophthalmic Symptoms in the Diagnosis and Localization of Brain Tumors," University of Sheffield, January 8; "The Importance of the Previous History and the Spontaneous Remarks of the Patient in the Diagnosis of Diseases of the Eye," County Hospital, York, January 9.

PERSONAL

Dr. Peter C. Kronfeld, formerly of Professor Meller's clinic in Vienna and for the past five years associate professor of ophthalmology in the University of Chicago, has been appointed professor and head of the Department of Ophthalmology of the Peiping Union Medical College, Peiping, China, and will assume his duties there about Jan. 1, 1934.

Correspondence

"Actinomycosis of the Orbit"

To the Editor:—In my article, entitled, "Actinomycosis of the Orbit" in the November, 1933, issue of the ARCHIVES, page 664, no reference was made to the interesting report on this subject presented by Dr. George A. Leahey at the convention of the American Ophthalmological Society in 1932. This was because volume 30 of the Transactions of the American Ophthalmological Society, in which Dr. Leahey's thesis can be found on page 457, became available, at least at the Barlow Medical Library of Los Angeles, only in April, 1933, when my paper was completed for publication.

M. N. BEIGELMAN, M.D., Los Angeles.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE HYALOID MEMBRANE OF THE VITREOUS. A. COWAN and W. E. FRY, Am. J. Ophth. **15:** 428 (May) 1932.

The authors refer to a previous paper in which they described a membrane in back of the lens revealed by the biomicroscope following intracapsular extraction. The present paper offers anatomic and histologic evidence of the presence of this membrane. The work was done on pig, monkey, kangaroo and human eyes, all of which were normal except the last named. Gross and microscopic preparations were made. In the former, after fixation and hardening of pig, monkey and kangaroo eyes, the cornea was removed at the limbus and the iris cut off at its base. The anterior portion of the suspensory ligament was severed, and the lens was extracted in its capsule. Then the posterior portion of the sclera with the retina was cut away at a point about 1 mm. behind the ora serrata. Next, under a dissecting microscope, the vitreous was carefully removed. This left a membrane-like structure having its attachment in the pars plana of the ciliary body which completely separated the aqueous from the vitreous chamber. The same membrane was demonstrated in microscopic sections of two pig and two human eyes. A number of illustrations are given.

W. S. REESE.

THE ORIGIN AND NATURE OF THE VITREOUS. Jose Pérez Llorca, Arch. de oftal. hispano-am. **32:** 540 (Oct.) 1932.

This is an exhaustive study of the origin and nature of the vitreous based on a histologic study of embryos of chicks and of some mammalia; it is illustrated by a large number of photomicrographs and black and white and colored drawings. The author's conclusions are as follows: 1. The vitreous is not a tissue, since it lacks a real structure. 2. It is not a secretory product of the ocular epithelium. 3. It seems to be a transudate, more or less modified by unknown circumstances. 4. Although there is some transudation through the epithelial layers of the fetal eye, it would seem that the major part takes place through the walls of the hyaloid vessels or of the pectensclera (in birds). 5. In later stages when the vitreous has become vascular, some participation may occur from the mesodermic tissue closing the fetal fissure. 6. Even though some participation by the ocular epithelium is admitted the exclusive ectodermic origin of the vitreous is inadmissible. The author gives a complete review of the different theories propounded to explain the origin of the vitreous.

C. E. FINLAY.

Bacteriology and Serology

THE BACTERIAL FLORA IN THE CONJUNCTIVAL SAC AND INTRA-OCULAR OPERATIONS. B. VON PELLATHY, Klin. Monatsbl. f. Augenh. **89:** 108 (July) 1932.

The author reported, and demonstrated tables, on the bacteriologic examination of 1,912 eyes prior to cataract extractions. He used blood agar, Loeffler's serum and bouillon. Staphylococcus albus and citreus were found in 65.3 per cent of the cases and Staph. aureus in 2.89 per cent; the pneumococcus in 17.88 per cent of senile cataracts and 44.78 per cent of congenital cataracts. Bacillus xerosis occurred in 51.1 per cent, Morax-Axenfeld's diplobacillus in 9.51 per cent and the influenza bacillus in 8.26 per cent. Bacillus fluorescens, although found in only 1.46 per cent of the cases, presented especial virulence. Pellathy experimented in vitro to find whether the various tissues of the eye offer especial advantages for the development of the various organisms. Staph. aureus thrived in all parts of the globe, but best on the cortical substance. Pneumococci remained active the longest on the cortical substance and in the aqueous humor; they disappeared everywhere within eight days. B. influenzae and Morax-Axenfeld's bacillus lost their virulence soon, but B. pyocyaneus and B. fluorescens were active after from ten to fourteen days. The pneumococcus, inoculated into the anterior chamber of rabbits, seemed to be only two and a half times more pathogenic than Staph. aureus.

K. L. STOLL.

Color Sense

CHROMATIC SENSE IN INDIRECT VISION. G. ZANETTIN, Ann. di ottal. e clin. ocul. **60:** 873 (Dec.) 1932.

Previous investigations have resulted in disagreement on such points as the extent of the color field in varying illumination and the relation of color to form sense. The effect of dark adaptation on the macular and extramacular color sense has not been thoroughly investigated.

The author employed an instrument devised by Lo Cascio, with which the smallest amounts of monochromatic light that gave color sensation when mixed with white light could be estimated for various zones of the retinal periphery. In ten normal subjects the four principal meridians were investigated at intervals of 10 degrees from 0 to 50 degrees on the perimeter, the chromatic acuity being expressed as a fraction of the macular acuity. There were individual variations, but in general there was a rapid fall in chromatic acuity outside of the 10 degree circle, more marked for red and green than for blue.

Thus at 10 degrees, chromatic perception for blue was 1/1.65 of that for the macula; for red, 1/2.7, and for green, 1/2.5. At 20 degrees, vision for blue was 1/3.3; for red and green, 1/7. In the temporal sector the reduction was more rapid than in the nasal sector, especially between 10 and 30 degrees. The extent of field in which the colors were perceived with the apparatus was greater for blue. Macular vision was greater for red than for blue and green, however. The subjects were examined after dark adaptation for fifteen minutes. A bibliography and tables are included.

S. R. GIFFORD.

BLUE-YELLOW SENSE OF PROTANOPES AND DEUTERANOPES COMPARED WITH THAT OF NORMAL TRICHROMATS. H. MEYER, Klin. Monatsbl. f. Augenh. **88:** 496 (April) 1932.

The differences of perception between protanopes (red blind) and deuteranopes (green blind) are of a physiologic nature, but not of physical origin, as is the absorption of light by the media. The experimental results of Liebermann and Marx were supported by Hess and Engelking, who found inferior blue-yellow perception in the red-blind, hypersensitiveness to blue-yellow in green-blind persons and a faint perception for these two colors in persons with total color blindness. Meyer employed three methods at the Eye Clinic at Freiburg to test the blue-yellow sense: by determining the field for colors, by finding the boundaries of minimum color perception with the graduated circular color scheme and by measuring the latitude of the neutral area in the spectrum. The blue-yellow sense of protanopic and protanomalous persons was found to be less acute than that of deuteranopes and normal trichromats. It is uncertain if this result, which coincides with the findings of Liebermann, Marx, Hess and Engelking, is present in every case. No hypersensitiveness for blue and yellow, however, was found in deuteranopes, as compared with normal trichromats, although it may happen, and although it seems to be superior to that of protanopic and protanomalous persons. Yet, it is impossible to decide whether the highest forms of normal color sense for blue-yellow of protanopic and protanomalous persons are inferior to that of deuteranopes.

K. L. STOLL.

Conjunctiva

FOLLICULAR (SWIMMING-POOL) CONJUNCTIVITIS. V. MORAX, Rev. internat. du trachome **9:** 8 (Jan.) 1932.

Though swimming-pool conjunctivitis can be otherwise transmitted, the swimming pool favors the spread of this type of infection. Typical inclusion bodies are occasionally found as in several other non-trachomatous conditions. Experimentally, when the conjunctival secretion of an infected patient is transferred to a healthy eye of another person, typical swimming-pool conjunctivitis develops after an incubation period of a few days.

J. E. LEBENSOHN.

STATISTICAL STUDY ON TRACHOMA IN THE BRANCH SCHOOLS OF THE PEIPING NORMAL UNIVERSITY. S. P. CHANG, Nat. M. J. China **18:** 958 (Oct.) 1932.

Among 2,528 students examined, 456 were found to be suffering from trachoma, that is, 12 per cent. Among 1,247 males examined, 19.7 per cent suffered from trachoma, while among 1,281 females, the percentage was 16.4. The percentage in regard to age showed that the chances of contagion increased with age. Under 10 years, the percentage was 7.2, while above 10 years, it was 28.9. Of the forms of trachoma, the papillary was predominant, and the cicatricial least seen.

S. H. McKEE.

HISTOLOGIC STUDIES ON MELANIN PIGMENT IN THE CONJUNCTIVA: II. PIGMENTATION OF THE CONJUNCTIVA FOLLOWING DISEASES OF VITAMIN A DEFECT. H. MATSUOKA, Acta Jap. Ophth. Soc. **36:** 131 (Nov.) 1932.

There can be no question but that dirty brown discoloration of the conjunctiva occurs in xerosis of the conjunctiva, though the cause for this discoloration is not known. The author examined five cases of conjunctival xerosis and found that the dirty brown discoloration is due to an increase of the melanin pigment in the epithelial layer of the conjunctiva. In this layer there are two kinds of increased pigmentation: The pigment occurs as a cap to the cell, or in the form of dendrites. The latter can be divided into two forms; in one the pigment is deposited in intracellular fissures, and in the other, in the form of cells with pigmented prolongations. The former is found in the superficial and middle layers of the epithelium. The other occurs in the deeper layers, while the prolongations pass upward to the superficial layers. In the middle and superficial layers, and in the basal layer, the pigment occurs in the form of intracellular pigment caps and around the nucleus. This pigmentation of the conjunctiva is an early symptom of the diseases due to a deficiency in vitamin A. It increases with the advance of the xerosis, and gradually disappears when the disease has improved. In the subconjunctival layer the increased pigment is found in the chromatophores and histiocytes, and there is a little pigment outside of the cells. There is no connection between these cells and the pigmented basal cells, though the prolongation of the dendrites may appear in the subepithelial tissue. The pigment in this vitamin A defect is epithelial, and the increase in the pigment results from abnormal metabolism of the epithelial cells.

A. KNAPP.

Congenital Anomalies

A PECULIAR MALFORMATION OF THE PAPILLA AND RETINA. F. GABARDI, Rassegna ital. d'ottal. **2:** 117 (Jan.-Feb.) 1933.

The literature of congenital malformations of the disk is reviewed and a table is given showing all the previously reported cases resembling that of the author. His case was that of a woman, aged 24, with vision reduced to ability to count fingers or less in each eye. The optic disks formed white masses, with indistinct outlines, from which the vessels emerged. White cords extended from these masses to the surrounding retina, the lower half of which was elevated in a number of folds. Other congenital defects were a marked dolichocephalism and facial asymmetry, and ectopia of the irides and lenses. Previous inflammatory conditions could apparently be excluded, and the author concludes that the findings are to be explained as a congenital defect due to abnormal proliferation of the mesenchyme owing to failure in closure of the fetal cleft. A bibliography is included.

S. R. GIFFORD.

Cornea and Sclera

AN UNUSUAL CORNEAL LESION PROBABLY CONGENITAL AND FAMILIAL IN CHARACTER. G. E. DE SCHWEINITZ and A. COWAN, Am. J. Ophth. **16:** 7 (Jan.) 1933.

A man, aged 48, exhibited numerous dotlike lesions in each cornea. These lesions were distributed over the entire cornea but much more densely in the center. The biomicroscope showed them to be in or immediately beneath Bowman's membrane. By direct illumination they appeared to be well defined, solid gray, plaquelike spots of various sizes and shapes, the largest measuring about 0.08 mm. across. The corneal epithelium was not involved and did not stain with fluorescein. The patient's son and daughter exhibited similar corneal disturbances. All had fairly high refractive errors, and the daughter had a covergent squint. The man's wife and another son did not have such lesions. The case of a man, aged 68, who presented a similar condition is also reported. The vision of all these persons was corrected to normal, except in the daughter with squint and in one eye of the father, in which vision of 6/7.5 was obtained. The authors consider the condition to be congenital and probably familial.

W. S. REESE.

KERATITIS NUMMULARIS (DIMMER). O. AUST, Arch. f. Ophth. **129:** 576 (March) 1933.

This corneal disease was first described by Dimmer in 1905 (four cases). In 1928 Salzmann reported his findings in fifty cases of this kind. Aust saw twenty-seven cases between Jan. 1, 1925, and Dec. 31, 1931. From these figures it does not look like a rare condition. It rather seems as though it had been overlooked or had not been correctly diagnosed.

The condition occurs almost exclusively among farmers. The most frequent age of the patient is between 15 and 30 years. The seasonal frequency curve shows the minimum incidence in April and May, and the maximum during August and September. The onset is sudden with tearing and photophobia which persists for from four to six weeks, at which time the patient usually notices a loss in visual acuity. At that time most of the patients consult an ophthalmologist who finds a pale eye with from ten to twenty small round corneal lesions of the same size (from 1 to 1.5 mm. in diameter).

Because of the similarity of these lesions to coins, i. e., *nummi,* the condition was called keratitis nummularis. The lesions are located in the anterior third of the cornea, usually some distance from the limbus, and have the characteristics of infiltrates in the anterior stroma. Their most peculiar feature is that after several months most of them develop into scars with definite flattening of the overlying surface (*Facetten*). Formation of ulcers is rare, according to Aust. The condition is nearly always confined to one eye. In two cases the second eye became affected several months after the first eye.

Histologic studies of fresh lesions removed for therapeutic reasons showed hardly any infiltrating cells. Keratoblasts and fragments of corneal lamellae seemed to make up the lesions. No treatment of any

kind (irradiation was apparently not tried) seemed to alter the course of the disease. The author had the best results with an iodine ointment and a constant bandage. No definite information concerning the possible cause has been obtained so far. The author thinks of the possibility of an ectogenic bacterial disease, the virus of which may be a plant or grain parasite.

P. C. KRONFELD.

Experimental Pathology

OCULAR LESIONS IN EXPERIMENTAL AVITAMINOSIS. L. LUGLI, Rassegna ital. d'ottal. **2:** 43 (Jan.-Feb.) 1933.

To exclude the factors of bacterial infection and drying of the corneal surface the author performed complete tarsorrhaphy on one eye in a group of white rats and then put the animals on a diet free from vitamin A. In only five of twenty-six animals did the lids remain completely closed during the experiment. In these animals no gross evidence of xerophthalmia occurred, and the conjunctiva was free from secretion on the operative side, while the other eyes showed marked keratomalacia, usually with perforation of the cornea. The closed eyes showed, however, a diffuse opacity of the corneal stroma on slit-lamp examination and signs of uveal inflammation. In eyes which showed a small opening advanced xerophthalmia developed, and the author believes that this indicates the importance of bacteria in producing the more obvious complications of the condition. The essential changes, however, in the epithelium and deeper structures are independent of bacteria and drying, as they appeared in the eyes protected by complete tarsorrhaphy.

S. R. GIFFORD.

EFFECT OF LONG-WAVED INFRA-RED RAYS ON THE RABBIT'S EYE. K. JOSURAN, Arch. f. Ophth. **129:** 353 (Jan.) 1933.

Only large quantities of nonpenetrating (long-waved) infra-red rays are harmful to the eye. Their effects are superficial burns of the cornea and conjunctiva.

P. C. KRONFELD.

REACTION OF RABBITS WITH SYPHILIS OF DIFFERENT STAGES TO ONE SINGLE INTRACORNEAL INJECTION OF FOREIGN SERUM. K. VOM HOFE, Klin. Monatsbl. f. Augenh. **89:** 28 (July) 1932.

The author found that rabbits infected with the spirochete of syphilis reacted to a single intracorneal injection of from 0.05 to 0.1 cc. of horse serum more frequently than normal animals. More severe irritation and denser, more widespread corneal opacities resulted in syphilitic animals. A positive reaction was obtained in five of eight normal rabbits, in two of four animals infected from two to three weeks previously and in eleven rabbits infected from five weeks to twelve months previously; one of the latter reacted instantly. The reaction was considered positive when spirochetes were found in the initial lesion microscopically and by inoculation of other animals.

K. L. STOLL.

TRANSPLANTATION OF RABBIT SARCOMA TO THE EYE OF A RABBIT AND TREATMENT OF THIS TUMOR WITH X-RAYS. M. YAMAMOTO, Acta Jap. Ophth. Soc. **36**: 147 (Dec.) 1932.

On transplanting the spindle cell sarcoma of Kato to the eyelid, Tenon's capsule, the lacrimal gland and other external parts of the eye, the tumor cells proliferate from the site of the transplantation, and, at the junction of the sclera and cornea, extend into the interior of the eye. After transplantation in the anterior chamber, the vitreous, the choroid, and other parts in the interior of the eye, the tumor extended into the adjacent tissue and then perforated the eyeball between the sclera and the cornea and continued to grow on the outside of the eye. Both forms are followed after a number of days by metastases in the lungs, liver and kidneys. Roentgen treatment is shown to be more efficient in repeated small doses than in a single large dose. It is important to determine what this fractional dose should be, and how frequently it should be repeated. The author obtained the best results with fractional doses of 300 roentgens given once every day.

A. KNAPP.

General

SOME EARLY OCULAR SYMPTOMS OF OVERINDULGENCE IN SUGAR AND SWEET FARINACEOUS FOOD. A. MAITLAND RAMSAY, Brit. M. J. 1: 266 (Feb. 18) 1933.

The function of an organ depends, to a great extent, on the size of the capillaries and the permeability of their walls to colloids. Transparency of tissue, which is so important in ocular structures, is facilitated, according to Duke-Elder, by the relative impermeability of the ocular capillaries to the passage of colloids. The action of metabolic toxins causes dilatation of the capillaries and increased permeability of their walls to the passage of colloids. Ramsay believes that one of the early clinical signs of this form of toxemia is a diminution in the translucency of the ophthalmoscopic picture. Translucency is the most delicate quality of the normal fundus oculi, and any loss of this quality is the first sign of ill health.

There is a group of patients suffering from headache who do not obtain the relief expected from the use of glasses. Examination of the eyegrounds shows slight changes in color and translucency. The light sense is always more or less disturbed. Headache is almost invariably present in the morning on first awakening from sleep, and examination of the urine shows further evidence of metabolic disturbance. These patients improve more rapidly when the intake of sugar and carbohydrates is restricted. As a result of this general treatment, in addition to the proper glasses, the eyegrounds rapidly regain their normal color and translucency, the visual symptoms disappear and headache is relieved.

Another example of nutritional disturbance is to be found in cases of phlyctenular conjunctivitis, which is always due to overindulgence in sweet starchy foodstuffs. There are, of course, other aggravating factors, like a lack of fresh air and an insanitary environment. The child suffers from impaired digestion and defective assimilation. Photophobia is one of the earliest and most characteristic symptoms. This is due to

spasmodic contraction of the circular muscle of the pupil, and the photophobia continues until the spasm is relieved. The mechanism of the production of photophobia is just as much a spasm of an unstriped muscle as that observed in the hollow viscera. In the treatment of these conditions the constitutional cause must be remedied. It consists in a chronic nutritional disorder due to derangement of the carbohydrate metabolism. The complications and sequelae of phlyctenular conjunctivitis are due to secondary infection.

Adults who suffer from toxic amblyopia, the result of overindulgence in tobacco or alcohol or both, generally suffer from a disturbance of carbohydrate metabolism; thus the systematic estimation of the blood sugar is an important point. Ramsay suggests that the full investigation of this subject offers a promising field for research.

A. KNAPP.

VITILIGO FROM THE REPEATED INSTILLATION OF ATROPINE. M. CORRADO, Ann. di ottal. e clin. ocul. **60:** 914 (Dec.) 1932.

In the author's first case, that of a girl 9 years of age, atropine had presumably been used for corneal ulcer secondary to trachoma at the age of 6. During this treatment the parents noted a loss of pigment involving the left upper lid, brow and temporal region, the hair and cilia becoming white. This lasted about twenty days, and when treatment was discontinued the normal color began to return. When the patient was seen by the author only the outer part of the lids and a small area of skin temporal to this remained white. The iris was of normal color. In a second case the author observed the occurrence of the same phenomenon during the use of atropine following an iridectomy for adherent leukoma. The patient was a man, 55 years of age, who showed a marked conjunctival intolerance to atropine, requiring its discontinuance. During use of this drug for twenty-five days the border, cilia and skin of the lids became gray and then almost white. After the drug was replaced by scopolamine hydrobromide the white area became darker again, but was still distinctly lighter than the surrounding skin on the patient's dismissal several weeks later. Notes are given concerning a case observed by Professor Maggiore, in which a child, sensitive to atropine, showed a similar vitiligo and a return to normal color when use of the drug was stopped.

The author supposes a predisposing condition of the skin in these cases which made it susceptible to the peculiar trophic disturbance following the use of atropine.

S. R. GIFFORD.

THE SPECIAL FEATURES OF OPHTHALMIC PRACTICE IN TROPICAL COUNTRIES. R. H. ELLIOT, Folia ophth. orient. **1:** 26, 1932.

Tropical ophthalmology has characteristics of its own. The ophthalmologist must familiarize himself with the language and customs of the people in order not only to facilitate a more sympathetic approach, but to understand the effects of the numerous domestic remedies. It is necessary to keep constantly in mind the part played by parasites and insects and the effect of climate on these and on bacterial growth. The amount of material gives unrivaled opportunity for practice and research.

G. M. BRUCE.

CHANGES IN THE OCULAR TISSUES CAUSED BY PARAPHENYLENDIAMINE. T. SANO, Acta Jap. Ophth. Soc. **36:** 107 (Aug.) 1932.

The author states that the action of paraphenylendiamine, which is used as a hair dye, can produce serious damage to any tissue of the body. He injected 0.7 or 1 per cent solution of this drug subcutaneously into white rats, 1 cc. to each 100 Gm. of body weight, and the eyes were examined six and twenty-four hours later. The agent produces marked hyperemia and edema in all of the ocular tissues. The exophthalmos depends on the swelling of the orbital tissue. The retina and its visual cell layer are particularly affected, while the cornea is less affected than the other tissues of the eye.

A. KNAPP.

General Diseases

A CONTRIBUTION TO THE STUDY OF DARK ADAPTATION IN NIGHT-BLINDNESS CAUSED BY AVITAMINOSIS. P. S. SOUDAKOFF, Nat. M. J. China **18:** 960 (Oct.) 1932.

Eight patients with vitamin A deficiency were studied in regard to the change in the primary and secondary dark-adaptation during the course of the illness. Both primary and secondary adaptation were found to be lowered in all the eight cases and in five to be greatly reduced. On admission, the primary adaptation ranged between 1/9 and 1/800, whereas the secondary adaptation taken thirty minutes after seclusion in a dark room ranged between 1/9 and 1/20,000. The decrease in primary and secondary dark-adaptation was more marked than that found in similar cases in Western countries. The lower the primary adaptation, the more defective was the secondary adaptation. Repeated examinations of these patients at intervals of from three to five days showed a gradual increase of sensitiveness to light, both initial and final. The curve of adaptation was Birch-Hirschfeld's type III. The type of the curve of the secondary adaptation in vitamin A deficiency seems to depend exclusively on the degree of involvement of the neuro-epithelial layer.

S. H. MCKEE.

EDEMA OF THE LIDS, CROUPOUS CONJUNCTIVITIS, ANGINA AND SORE THROAT AND SWELLING OF THE REGIONAL LYMPH GLANDS. A. LOWENSTEIN, Med. Klin. **29:** 9 (Jan. 1) 1933.

The author reports an unusual case in which swelling of the lids was noted first, then croupous conjunctivitis; angina with a sore throat developed next, and then swelling of the submaxillary, preauricular and lingual lymph glands accompanied by localized headache. The injection of serum obtained from the swollen lid into a rabbit's cornea resulted in severe keratoconjunctivitis with herpetic spots. Three rabbits treated in this manner died of herpetic encephalitis. A similar result was noted when secretion from the tonsil was injected into a rabbit. The author traces the course of the virus from the lid, through the conjunctiva, down the lacrimal passages to the throat and then to the regional lymph glands. He thinks it is likely that the headache was due to localized herpes encephalitis of the brain.

L. L. MAYER.

General Pathology

OCULAR CHANGES IN LEPROSY: HISTOLOGIC OBSERVATIONS. A. SANTONASTASO, Ann. di ottal. e clin. ocul. **60:** 796 (Nov.) 1932.

The author sums up the conclusions from data reported in the previous instalments of his extremely thorough study. He describes the conjunctival changes from small infiltrations at the limbus to large destructive lesions involving the sclera and cornea. The infiltration surrounds the vessels in a characteristic manner, is rich in plasma cells and contains numerous organisms. The fatty degeneration described in previous reports is present in these lesions. Foci containing numerous bacilli were found in the corneal parenchyma at some distance from the vessels. The histologic picture varied considerably in various corneas.

In the ciliary body a characteristic change was loss of pigment from the epithelium. The ciliary body, iris and choroid showed marked atrophy in many cases. Changes in the ciliary body were always continuous with similar changes in the episcleral tissues.

The author concludes that the globe is invaded chiefly by direct penetration of bacilli through the conjunctival and corneal epithelium. He thinks that infection may occur by the endogenous route, but that this is rare. In general, the infiltration was most intense in those parts of the eye in which the reticulo-endothelial system was best developed, and the author insists on the importance of this relation in the histologic picture of leprosy. Twenty-three photomicrographs are shown.

S. R. GIFFORD.

PATHOLOGIC HISTOLOGY OF THE EYE IN ECLAMPTIC AMAUROSIS. M. UYEMURA, Acta Jap. Ophth. Soc. **36:** 92 (July) 1932.

The author found the principal ophthalmoscopic changes in eclamptic amaurosis to be: (1) narrowing of the retinal arteries, and (2) edematous clouding of the disk and retina. He examined the eyes of a pregnant woman who died in an eclamptic attack after suffering from eclamptic amaurosis. In the choroid he observed a breaking up of the vascular walls and hyaloid degeneration of the capillaries, resulting in obliteration of the lumen and many small hemorrhages. The retina presented a serous detachment at the posterior pole; there were edema, vacuoles and hemorrhages in all of the layers, while the vessels themselves were intact. The optic nerve showed a breaking up of the walls of the small vessels.

A. KNAPP.

Glaucoma

CONGENITAL HYDROPHTHALMOS: ANATOMIC ALTERATIONS. E. KALT, Ann. d'ocul. **170:** 97 (Feb.) 1933.

Infantile hydrophthalmos in perhaps a majority of cases is the result of an infectious lesion of the vascular system of the eye: the central vessels of the optic nerve, the anterior and posterior ciliary vessels, the vorticose veins and the choroidal vessels. The slowly developing changes have only one result. The intra-ocular hypertension might be explained, in the first place, as the effect on the intra-ocular vasomotor system of toxic substances emanating from the affected ves-

sels or resulting from nonelimination; it might also be the result of condensation, atrophy of the uveal tract or blocking of the canal of Schlemm.

S. H. McKee.

Clinical Observations on Eyes with Fistulas. F. Poos, Arch. f. Augenh. **107:** 163, 1933.

It is generally recognized that the tension in an eye which has a permanent fistula is regulated by a number of factors, chief among which are the rate of formation of fluid and the resistance offered to the escape of fluid by the tissues covering the fistula, i. e., the conjunctival scar. In addition to these factors, however, there are others which have received scant investigation, and it is to these that the author calls attention.

Two cases are reported in which fistulas were present in nonglaucomatous, healthy eyes. The first case was that of a man who had been seen and reported on four years previously by Rosenthal (*Klin. Monatsbl. f. Augenh.* **82:** 88, 1929). One eye, which was healthy in all other respects, had a fistula, apparently from some congenital anomaly, at the temporal limbus, at about 9 o'clock. The second case was that of a girl who had had a penetrating wound of the sclera at the limbus in the center of the palpebral fissure; the eye was healthy and normal with the exception of the fistula and an operative coloboma of the iris.

Careful repeated observations of the tension in these two cases showed a characteristic curve for the day. On arising in the morning the tension was always elevated, but it soon began to drop, reaching a low level in the evening. The tension ranged from 22 to 7 mm. of mercury. The cause of the increase in tension was apparently a closing off of the fistula by the lids covering the eyes at night.

In the case of congenital fistula, in which iridectomy had not been performed, the effect of the size of the pupil on the tension could easily be demonstrated by means of mydriatics or by changing the size of the pupil through different degrees of illumination. By means of Seidel's fluorescein test the author was able to demonstrate a decrease in outflow from the anterior chamber when the pupil was dilated with atropine.

He was able to confirm these findings in two cases of chronic simple glaucoma in which Elliot trephination had been performed. In both cases closing the lids raised the pressure, but when the upper lids were elevated away from the trephine opening and the patient was directed to look down, the tension gradually sank.

Attention is called to the fact that although the tension in one case was considerably below normal there was a progressive increase in myopia throughout the period of school attendance.

F. H. Adler.

Glaucoma: Treatment of Early Cases. A. F. MacCallan, Folia ophth. orient. **2:** 156, 1933.

This article commences with the report of a case of simple glaucoma in which there were characteristic symptoms of the disease, but in which the only definite sign was great enlargement of one blind spot. Bilateral trephining was done, with good visual results and relief from all symptoms.

The author believes that both eyes should be operated on at the first sign of glaucoma in either one, whether tension can be controlled by miotics or not. He believes that the vision can be maintained at the preoperation level by this means, whereas delay usually causes the loss of a test line. Often when one eye is operated on, the deterioration of the other eye is not observed by the patient until too late. Increase of the cupping, atrophy, and subsequent cataract formation are not likely to occur after early decompression.

G. M. BRUCE.

Hygiene, Sociology, Education and History

FIFTY YEARS OF OPHTHALMOLOGIC PROGRESS IN EGYPT. M. MEYERHOF, Folia ophth. orient. **1**: 5, 1932.

Although ocular disease and blindness have been rife in Egypt for ages, the problem was not sufficiently attacked until comparatively recent years. As late as 1882, there were only five oculists and one ocular department in a general hospital in all Egypt. In 1903, real progress began with the endowment by Sir Ernest Cassel of two traveling ophthalmic hospitals. The chief of this service was MacCallan. By the time of his retirement in 1924, he had extended the ophthalmic organization to include twenty permanent and five traveling hospitals. His pupils carried on his work, and by 1930 there were forty-six ophthalmic hospitals with 1,100 beds. The number of patients treated in that year was 526,000, and the operations performed numbered 200,000. The free institutions are now entirely in the hands of national specialists. Quackery, a curse of former days, has been repressed to a remarkable degree, and blindness has been reduced to 1.7 per cent among the patients of the ophthalmic hospitals.

G. M. BRUCE.

IRIDOCYCLITIS FOLLOWING THE STING OF A WASP. T. OTSUKA, Acta Jap. Ophth. Soc. **36**: 105 (Aug.) 1932.

A man, aged 44, received the sting of a wasp in his left eye. The very severe iridocyclitis which followed led to enucleation after six weeks. The sting of the wasp had perforated the cornea and thus entered the anterior chamber, leading to necrotic inflammation of all the layers of the cornea with fibrinous exudation in the anterior chamber, necrosis of the endothelium, total posterior synechia with organized exudates on the surface of the lens and hyaline degeneration and endothelial proliferation of the vessels of the iris, with numerous hemorrhages on the surface. The ciliary body was severely affected. The sclera was thickened with fibrinous exudates. The vitreous presented old blood which was partly organized. There was an area of old hemorrhage and exudate between the retina and the choroid in the posterior segment. The nerve head was swollen and projected into the vitreous with perivasculitis of the vessels.

A. KNAPP.

Injuries

TWO CASES OF INJURIES TO THE EYES FROM COAL GAS POISONING. M. UYEMURA, Acta Jap. Ophth. Soc. **36:** 134 (Nov.) 1932.

Two brothers slept in the same room with a coal gas oven. On the following morning they were found asphyxiated, and the room was filled with gas. After five hours, they recovered. During the following four days they complained of severe headache and nausea, and the older brother had trouble with his sight. On the seventh day, the following condition was found:

The color of the skin of the older brother was somewhat pale. The reflexes were normal. There was no disturbance in the movement of the eyes. The pupils were very small. There was prompt reaction. Vision was 1/200. The optic disks were red, cloudy and swollen (2 diopters). The region about the disk was a grayish white. The vessels were normal. After one month vision returned, and the changes in the eyeground had disappeared.

The general symptoms of the younger brother were the same. The pupils were very small. There was a prompt reaction. The eyegrounds were normal. On the twelfth day, there was a slight muscular paralysis of the right eye, which disappeared after one day. A. KNAPP.

Lens

THE CALCIUM CONTENT OF NORMAL AND PATHOLOGIC LENSES. P. GRABAR and J. NORDMANN, Compt. rend. Soc. de biol. **112:** 1534, 1933.

Normal lenses from persons from 6 months to 78 years of age vary little in their calcium content, containing usually about 2.5 mg. per hundred grams of fresh lens. In senile cataract, on the other hand, the calcium content reaches from 27 to 60 mg. The increase in calcium may be so marked as to render the lens opaque to the x-rays. Since determinations of the serum calcium in cataractous patients show no significant deviation from normal, the accumulation of calcium in cataract must represent a purely local disturbance of metabolism.

J. E. LEBENSOHN.

BELATED ROSETTES OF THE LENS AFTER CONTUSION: RUDIMENTARY FORMS. H. SCHLÄPFER, Klin. Monatsbl. f. Augenh. **89:** 346 (Sept.) 1932.

The author renders a historical account of publications on late formations of rosettes of the lens after contusion, and reports seven cases from Vogt's clinic in Zürich. The rosettes developed either in complete form or in the shape of sectors or rudiments. The formations were distinctly limited to the same zone of discontinuity, and their traumatic origin was evident. The seams of the lens between the sectors frequently presented the form of a lobed leaf. The design of the fibers was partly blurred. A retracted sulcus with considerable thickening of the capsule was observed in one case. All the patients had suffered a severe contusion in early youth. It seems certain, therefore, that this

type of cataract occurs more frequently after contusions in young persons than in adults. It is not known if it occurs in advanced years of life. Traumatic changes of the iris were found in some cases in which a traumatic origin could not be established by the anamnesis.

K. L. STOLL.

Methods of Examination

FOREIGN BODIES OF THE CILIARY AND POSTCILIARY REGION LOCATED BY DIGITAL PRESSURE AND DIAPHANOSCOPY. A. TRANTAS, Folia ophth. orient. **1:** 61, 1932.

Lesions and foreign bodies lying in the extreme periphery of the fundus, in the ciliary body or in the angle of the anterior chamber can be visualized by pressing into the globe over the region by means of the finger or the transilluminator. The area so brought into view is examined with the ophthalmoscope or the microscope of Czapski. The description of the technic is clarified by the liberal use of photographs and illustrations.

G. M. BRUCE.

Neurology

CHANGES OF THE EYEGROUND IN WILSON'S DISEASE (PSEUDOSCLEROSIS). A. PILLAT, Am. J. Ophth. **16:** 1 (Jan.) 1933.

The author reviews the literature on pseudosclerosis and reports a typical case of Wilson's disease (pseudosclerosis Westphal-Strümpell) with a Kayser-Fleischer pigment ring in both corneas, and a rather peculiar and hitherto undescribed disease of the fundus. He gives the following summary:

"The fundus disease is characterized chiefly by two points: (1) the presence of white dots either isolated or conglomerated in the middle or outer layers of the retina, which increase in numbers towards the periphery, (2) a markedly diffuse degeneration in those parts of the fundus in which the white dots lie.

"The retinal vessels are unaltered. So also are the macula and the choroid.

"The two fundi are very differently affected, the right much more than the left.

"The presence of nightblindness in the more seriously affected eye points to a local degenerative process of the retina.

"Parallelism of changes in the various parts of the brain and of the eye is suggested.

"The differential diagnosis between this fundus disease and other fundus conditions associated with white dots in the middle and outer layers of the retina is briefly discussed."

W. S. REESE.

A TYPICAL CASE OF CHRONIC PROGRESSIVE OPHTHALMOPLEGIA EXTERNA. A. JUHASZ-SCHÄFFER, Klin. Monatsbl. f. Augenh. **89:** 59 (July) 1932.

The author discusses the literature to date, and reports a typical case of chronic progressive external ophthalmoplegia. A man, aged

71, had not been ill since his fortieth year; the family anamnesis was not significant, and no consanguinity existed. The Wassermann reaction of the blood and spinal fluid was negative. Ptosis of the upper lid of the left eye had been observed thirty years before, when the lid of the right eye drooped only slightly. The degree of the ptosis varied according to the amount of daily strain. He never had diplopia, which the author explains as the result of the chronic bilateral development of the disease. The lower lids of both eyes were limp, and showed slight ectropion; the upper lids could be raised only slightly and with effort. The globes were fixed and could not be moved more than from 2 to 4 degrees, the right in a temporal and downward direction and the left downward and to the nasal side. The pupils were round, and reacted promptly in every respect. Ability to converge was lacking. The onset of the ocular symptoms could not be determined. Aside from them no subjective disorders of sensitiveness or motility existed. The reflexes were normal; the sense of taste and the olfactory and acoustic nerves showed normal function. Lesions in the area of the pons, the medulla oblongata and the corpora quadrigemina and geniculata could not be detected. The patient presented isolated lesions of the nuclei of the third, fourth and sixth nerves. K. L. STOLL.

Operations

A RADICAL OPERATION FOR TRICHIASIS AND ENTROPION OF THE UPPER LID. P. CHRONIS, Folia ophth. orient. **2**: 187, 1933.

This operation is a further modification of the Hotz-Streatfield-Snellen group of procedures. The technic is summarized by the author as follows.

First step: Canthoplasty is performed. Second step: The Jaeger plate is introduced, and, parallel to the palpebral border, from 2 to 3 mm. from the cilia, an incision is made extending from the inner portion to the external angle, which must not affect more than the skin; the latter is lifted up in order to expose the palpebral muscle. Third step: All the fibers of the palpebral muscle are seized and cut out in order to expose the tarsus. After removal of the palpebral muscle, the small muscle of Horner, which covers the roots of the cilia, is also removed. Fourth step: With a narrow knife thin slices are cut away from the hypertrophic tarsus by small successive cuts, this being necessary to make the tarsus thin and smooth. Fifth step: Sutures are inserted. On the whole, eight or nine sutures are needed, three for the canthoplasty and five or six for the lid. The sutures for the lid are inserted in the following manner: Starting at the inner angle, the needle is passed through the skin above the cilia from the outside to the inside without touching the roots, and then the sutures are passed horizontally through the fascia tarso-orbitalis. Insertion of the sutures of the canthoplasty is then done. Sixth step: The threads are tied. Seventh step: An intermarginal incision is made. The lid is stretched over the Jaeger plate, and with the point of the knife a marginal incision is made, approximately 1 mm. deep, from one end to the other, behind the cilia at the level of the white line separating the glands

from the meibomian orifices. The skin of the lid is left to heal by second intention. An aseptic dressing is applied. On the third day the threads are removed.

G. M. Bruce.

Orbit, Eyeball and Acessory Sinuses

Ten Cases of Operation for Intra-Ocular Cysticercus. G. G. Loginoff, Sovet. vestnik oftal. **2**: 5, 1933.

The author describes the operation for removal of a cysticercus. A meridional incision of the sclera was used after either the external or the internal rectus had been divided. In one case, in which the divided muscle was sutured, marked strabismus took place. He therefore left the muscle unsutured in all the other cases, and the cosmetic result was good. Iritis, which lasted from six to twenty-eight days, developed in six cases after the operation. In two cases connective tissue formed about the cysticercus which evidently prevented the iris from becoming inflamed. Prolapse of the vitreous occurred in three cases. The tension was below normal in only one case. The ophthalmoscope revealed the contour of the cysticercus and the greenish-yellow hue of its margin in five cases. In the others, in which reactive inflammation led to the formation of connective or granulation tissue, it was impossible to make out the outline of the parasite. The author advises not to operate in these cases, because the connective tissue forms adhesions and may not allow the vesicle to escape during the incision. The duration of observation was from one to five years. Vision improved from 1/10 to 7/10 in two cases and remained unchanged in the others.

In two cases endophthalmitis and iridocyclitis developed. Enucleation of these eyes was performed. The cysticercus of one enucleated eye was transplanted into the vitreous of a rabbit's eye. The pathologic findings in the two human eyes and the rabbit's eye are given by the author in detail. The uveal tract was affected markedly where the cysticercus was in contact with the tissue. There was also an accumulation of mononuclear eosinophils. The author believes that while the cysticercus is alive it acts as an aseptic foreign body, with slow development of a granuloma. The wandering cells participate in the formation of the granuloma, as does also the uveal tract by proliferation of its cells. In the later stage, either endophthalmitis develops, which is produced by the toxins liberated on the death of the cysticercus, or the process takes a course of proliferating inflammation and uveitis with typical signs of sympathetic ophthalmia may ensue.

O. Sitchevska.

Pharmacology

A New Anesthetic for Use in Ophthalmologic Surgery (*p*-n-Butylaminobenzoate of Dimethylaminoethanol). N. Shimkin, Folia ophth. orient. **2**: 192, 1933.

The author has carried on clinical and experimental research in the use of *p*-n-butylaminobenzoate of dimethylaminoethanol as a local anesthetic in operations on the eye. The drug is inexpensive, and since it is not habit-forming its sale is not restricted. It is soluble and remains

unchanged by frequent boiling. The 1 per cent solution, administered either by instillation or by injection, is superior to the local anesthetics now in vogue.

G. M. BRUCE.

Physiology

THE CIRCULATION OF THE INTRAOCULAR FLUID. C. BERENS and A. POSNER, Am. J. Ophth. **16:** 19 (Jan.) 1933.

The authors review the literature on experimental proof that the optic nerve serves as an outflow channel for the intra-ocular fluid. They experimented on animal and human eyes, using Weed and Wegefarth's prussian blue precipitation method. This consists of an injection of potassium ferrocyanide and iron ammonium citrate into the vitreous fifteen minutes before enucleation, following which the eye is fixed in a dilute solution of formaldehyde and 1 per cent hydrochloric acid so as to cause the injected material to be precipitated as prussian blue. Of the animals used, only the rabbit showed evidence of drainage of the intra-ocular fluid through the optic nerve and the posterior portion of the retina. Of the five human eyes used, one was normal and four were pathologic. In none of the latter were prussian blue granules found in the optic nerve. In the normal eye the granules were found arranged around the central vessels of the optic nerve for a distance of 1.5 mm. from the papilla. In this case the injection into the vitreous was made twenty-four minutes before enucleation. W. S. REESE.

THE ORIGIN OF INTRAOCULAR PRESSURE. S. SONDERMANN, Am. J. Ophth. **16:** 97 (Feb.) 1933.

The author decries the attempts to explain pathologically increased intra-ocular pressure without first explaining how normal intra-ocular pressure originates. He confirms Dieter's statement that intra-ocular pressure equals hydrostatic pressure minus colloidosmotic pressure. Heretofore the value for the blood pressure in the retinal veins was applied to the uveal veins also, and when the osmotic pressure was deducted there was nothing left for intra-ocular pressure. The author's tests show a much higher pressure in the vorticose than in the retinal veins; so Dieter's hypothesis becomes plausible. The author explains the marked difference between the pressure in the retinal and that in the uveal veins by developmental changes. Essentially the arterial blood enters the eye under the least possible obstructive pressure while the venous blood leaves the eye where the pressure is greatest.

W. S. REESE.

A NEW TECHNIC FOR DENERVATING THE EYE OF THE CAT. Z. M. BACQ and G. LEPLAT, Compt. rend. Soc. de biol. **112:** 1549,1933.

The removal of the superior cervical ganglion in the cat assures the postganglionic sympathetic denervation of the eye and renders the iris more susceptible to dilatation on the intravenous injection of epinephrine hydrochloride. If, after a week's interval, this procedure is followed by retrobulbar injections of procaine hydrochloride and 95

per cent alcohol, the ciliary innervation is also blocked and the eye is completely denervated. The pupil, at first markedly dilated, returns to normal size in twenty-four hours. With an eye so prepared, the passage in the blood of sympathomimetic substances can be critically tested.

J. E. LEBENSOHN.

Refraction and Accommodation

GENETICS OF REFRACTION. E. SCHWARZ, Arch. f. Ophth. **129:** 361 (Jan.) 1933.

On 479 rabbits of ages varying from over 100 days up to 5 years, the refraction of the corneas was determined by keratometry and the total refraction measured by retinoscopy under atropine (one hour after one instillation of a 1 per cent solution). The degrees of astigmatism and anisometropia were so slight that the average of the four main meridians of every animal was used as the unit of refraction. During the first year of life a definite negative correlation was found between corneal refraction and age; in other words, toward the end of the first year of life more animals with lower corneal refractions were found, and vice versa. The arithmetical means of corneal curvature for the whole series of rabbits was 44.25 diopters; the variations observed amounted to 17 diopters.

The total refraction varied from —1 to +6 diopters only; the means were +2.04 diopters for the whole series of rabbits, + 2.23 for the animals under 1 year of age, and +1.8 for those over 2 years of age. There was, however, no definite correlation between age and total refraction.

The study of the correlation between corneal and total refraction during the first year of life suggests that the change in total refraction which should result from the decreased corneal refraction is made up for by the longitudinal growth of the eyeball.

Definite conclusions concerning the hereditary factor in the individual refraction will be justified only if results of cross-experiments on a large scale are available.

P. C. KRONFELD.

CHANGES IN REFRACTION IN DIABETES. K. O. GRANSTRÖM, Acta ophth. (supp.), 1933, vol. 11.

A diabetic myopia occurs only in untreated diabetes—therefore at a time when the sugar in the blood is increasing. Hyperopia frequently occurs after the beginning of treatment and lasts from two to four weeks. Acetone bodies and acidosis have nothing to do with the development of either the myopia or the hyperopia. The radius of curvature of the cornea does not change. There is no change in the position of the macula, and the changes in the refractive index are found essentially in the axial part of the refracting system, while the refractive index of the periphery remains unchanged. The changes in refraction must depend on changes in the lens. These changes are intracapsular and apparently consist of an altered index or refraction of the central part of the lens. The observations on accommodation confirm this idea.

The author believes that the diabetic changes in refraction occur somewhat as follows:

In an untreated patient with diabetes there is a retention of salt in the body tissues. Granström assumes that this retention also occurs in the lens, especially in the nucleus, as a consequence of which its index of refraction is increased. This would explain the development of myopia. When the glycosuria diminishes the retained salt causes a transitory retention of fluid in the tissues. This retention of fluid in the nucleus of the lens causes a lower index of refraction, which produces the hyperopia.

F. H. Adler.

Retina and Optic Nerve

Changes of the Optic Nerve Resulting from Pressure of Arteriosclerotic Internal Carotid Arteries. O. Saphir, Am. J. Ophth. **16:** 110 (Feb.) 1933.

The author gives the following summary: "The literature on changes of the optic nerve resulting from pressure of arteriosclerotic internal carotid arteries is reviewed. The result of a study of six such cases is reported. The carotid arteries were markedly sclerosed and in four instances revealed an evident aneurysm. The optic nerves grossly were compressed, medially displaced, flattened and thinned out. Histologically, the changes were very slight and consisted of a moderate increase in glia and connective-tissue fibers and an occasional myelin-sheath degeneration affecting mainly the centrally located bundles. Gross and histologic lesions in the optic nerve were encountered only in those portions of the nerve extending between the chiasma and the optic canal. Because of the slight histological changes and in view of the fact that in none of the cases was an impairment of sight recognized clinically, though it is realized that slight defects in the visual field are often not noted by the patient, it is unlikely that visual disturbances, if present in old people, should often be explained by lesions in the optic nerves resulting from pressure of arteriosclerotic internal carotid arteries."

W. S. Reese.

Holes in Secondary Detachment of the Retina in Juvenile Emmetropes: Report of Cases. K. Velhagen, Jr., Klin. Monatsbl. f. Augenh. **88:** 317 (March) 1932.

Five emmetropic persons with detachment of the retina, between 20 and 32 years of age, presented no general pathologic changes, although the tuberculin reaction was positive in all of them; old tuberculin produced some general and local reactions. This experience would suggest a tuberculous origin of these retinal detachments, which, for this reason, were termed secondary. Tears, observed in only a few cases of secondary retinal detachment, were present in all five cases. The tears appeared to be fairly accessible; the vitreous and anterior portion of the globes were free from complications. The treatment, nevertheless, should be conservative and expectant in such cases, and surgical procedures should be resorted to in exceptional conditions only. The second of Velhagen's cases showed an arrangement of yellowish-white spots,

free from pigmentation, in the retina, resembling those in a case of retinitis tuberculosa observed by Arnold Knapp. Mention is made of retinal detachment with a tear in an emmetropic woman, aged 36, suffering from severe diabetes.

K. L. STOLL.

POLYCYTHEMIA AND EMBOLISM OF THE CENTRAL RETINAL ARTERY: REPORT OF CASES. A. ELSCHING and W. NONNENBRUCH, Klin. Monatsbl. f. Augenh. **88:** 433 (April) 1932.

Two cases of polycythemia and embolism of the central retinal artery are reported. The first patient, a man, aged 45, had noticed obscurations in both eyes for a number of years. They finally disappeared after treatments with roentgen rays. The second patient, aged 51, became suddenly blind during an attack of hemiplegia. Both patients had initial hyperglobulia of 6,500,000 red corpuscles; their spleens were not swollen. The blood pressure remained normal (135 mm.) during the treatment in the first case, but was increased (175 mm.) at first in the second case, which is classed in the group of hypertonic polyglobulia. The first case, presenting an uncertain etiology, did not belong to either one of the two types of polycythemia; some disturbance in the regulation center may have been caused by an injury which the man suffered during the World War. The connection between embolism of the central retinal artery and polycythemia is discussed, and a case reported by Mylius is quoted. The obscurations, especially the protracted ones observed in the first case, are explained as spasms in the ophthalmic or central retinal artery. Changes in the circulation of the blood, brought on by polycythemia, may support the occlusion of the blood vessels. The embolism in most of these cases is genuine.

K. L. STOLL.

DISCIFORM DEGENERATION OF THE MACULA LUTEA: REPORT OF A CASE. H. P. M. DUYNSTEE, Klin. Monatsbl. f. Augenh. **88:** 511 (April) 1932.

The anamnesis and general physical condition of a man, aged 60, showed no pathologic features aside from slight hypertension. Vision was 2/30 in the left eye and 5/5 in the right in 1928, when numerous yellowish-white dots were found in the macula, a few in the periphery of the left eye and four in the macula of the right. Two years later, typical disciform degeneration of the macula had developed in the left eye, in the periphery of which an increased number of yellowish dots and an accumulation of pigment were noted. The fundus of the right eye presented the appearance of the left two years previously; the dots disappeared soon afterward, but the entire region of the macula changed into a white, cloudy, floccular layer, above which the superior retinal layers seemed to be raised. New vessels, not connected with the retinal vessels, seemed to have formed in this area, and a fluid seemed to have formed beneath the retina. The degeneration of the macula of the right eye was complete two months later. Peripheral choroidal changes in connection with disciform degeneration in the macula were observed only in one case, which was described by Hanssen, who thought that there was no etiologic connection between them. Duynstee contradicts this opinion, because he observed their simultaneous development. Contrary to the view of Kuhnt and Junius, the author thinks that the disease

began in the choriocapillary layer, which nourishes the deepest layers of the retina, because the yellowish dots were located in them, while the retinal vessels appeared perfectly normal at first, and crossed the disk in a normal manner after the process had become stationary.

K. L. STOLL.

ETIOLOGY OF RETROBULBAR NEURITIS FROM THE STANDPOINT OF THE NEUROLOGIST. A. VON SARBÓ, Klin. Monatsbl. f. Augenh. **88:** 762 (June) 1932.

The author accepts Wilbrand and Sänger's division of retrobulbar optic neuritis into two groups. The first, called neuritis axialis, affects the papillomacular fibers and causes a central scotoma with intact peripheral vision. The second form, neuritis interstitialis peripherica, affects the marginal fibers of the optic nerve, causing concentric contraction of the visual field with good central vision and the normal field for colors. Von Sarbó discusses diseases of the posterior sinus and multiple sclerosis as etiologic factors.

Diseases of the retrobulbar segment of the optic nerve in acute infectious diseases are chiefly due to disorders of the nose and adnexa. Generally speaking, retrobulbar neuritis is caused more frequently by multiple sclerosis than by infections of the posterior sinuses.

The visual disturbance in multiple sclerosis begins suddenly and reaches its height rapidly; spontaneous improvement is the rule. A case of complete amaurosis has not been observed in more than six hundred cases. Characteristic undulations in the intensity of the disturbance of the optic nerve occur, leaving discoloration of the temporal halves of both disks; it is the final symptom of the pathologic process in the axial fibers of the optic nerve. This ascending degeneration develops slowly, so that functional disorders of the axial fibers may be manifested by the presence of a scotoma while the disk still appears normal. Pallor, usually bilateral in multiple sclerosis, may lead to an erroneous diagnosis, and the cause may be attributed to disease of the posterior sinus, if present in one eye only.

Even if only minor ocular disorders exist, an early diagnosis may be possible in the presence of a number of symptoms: paresthesias of the extremities and face, vertigo, dysfunction of the sense of taste, aphonia, sudden disturbance of speech and writing and retention of urine. For the differential diagnosis, the insertion of epinephrine tampons into the affected nostril and the colloid curve of the spinal fluid, which should be positive in multiple sclerosis and negative in rhinogenous disease, may be of assistance. The author observed several cases of undoubted multiple sclerosis which developed after influenza.

K. L. STOLL.

METABOLISM OF THE RETINA: IV. THE ACTION OF WOOD ALCOHOL, QUININE AND ILLUMINATING GAS ON THE METABOLISM OF THE RETINA. TAWADO OGUCHI, Acta Jap. Ophth. Soc. **36:** 155 (Dec.) 1932.

Wood alcohol, quinine and illuminating gas produce sudden loss of vision. Is metabolism of the retina influenced by these poisons?

The author's experiments show that these poisons influence metabolism of the retina:

1. Two cubic centimeters of 20 per cent solution of wood alcohol was injected into a frog 30 grains (1.95 Gm.) in weight, and after thirty minutes the Qo2 (30 C.) was reduced by 41 per cent.

2. After two injections of a 50 per cent solution of wood alcohol, 1 cc. per hundred grains (2,110.4 Gm.) of body weight, into a rat, the Qo2 (37.50 C.) was reduced by 50.2 per cent.

3. One cubic centimeter of 1 per cent solution of quinine was injected into a frog 30 grains in weight, and after thirty minutes the Qo2 (30 C.) was reduced by 21.3 per cent.

4. After two injections of 1 per cent solution of quinine, 1 cc. per hundred grains of body weight, into a rat, the Qo2 (37.5 C.) was reduced by 27.5 per cent.

5. After poisoning with illuminating gas in a rat, the Qo2 (37.50 C.) was reduced by 27 per cent.

A. KNAPP.

Trachoma

TREATMENT OF TRACHOMA WITH ETHYL BENZYL CINNAMATE. J. JACOBSON, Rev. internat. du trachome **10:** 100, 1933.

Daily intramuscular injections of 1 cc. of ethyl benzyl cinnamate are given for twelve days, followed by a two week rest period. Generally three series of treatments are given. The dose is reduced to 0.5 cc. for children between 5 and 10, and to 0.25 cc. for children under 5 years. The injections are painless and well tolerated, causing focal, but no local or general, reactions.

The subjective symptoms are the first to abate. Vasodilatation is noticed at first, but the congestion is unaccompanied by infiltration. The corneal infiltrations and pannus then regress, the xerotic glaze passes, the lids become more supple, and the papillary granulations tend to resorb. Such improvement was noted in 137 of 244 stubborn cases in which this treatment was used either exclusively or combined with local measures. The trachomatous lesions are attacked through the circulation.

J. E. LEBENSOHN.

THE ETIOLOGY OF TRACHOMA. A. RÖTTH, Arch. f. Ophth. **128:** 381 (June) 1932.

The inclusion bodies of Prowazek are still the subject of heated discussion. It is certain that they are not signs of cellular degeneration. They do not originate from normal constituents of the epithelium cell. Prowazek's bodies are found in almost every case of fresh trachoma and frequently in chronic trachoma. The author found them in 50 per cent of his patients who had trachoma for over five years. Prowazek's bodies are also found in a type of acute conjunctivitis caused by a virus of genital origin, the inclusion body blenorrhea of new-born infants or of adults. In this disease the Prowazek bodies can be demonstrated in 100 per cent of the eyes affected. The disease heals within a few weeks without any sequelae or a slight superficial cicatrization. In a few cases the formation of an epitarsus seems to follow this type of conjunctivitis.

Conjunctivitis contracted in a swimming pool is not a clinical or a bacteriologic entity. In some of the cases belonging in this group, Prowazek's bodies were found. The microscopic picture of these bodies and the negative cultures suggest strongly that they are living organisms, most likely of the group of Chlamydozoa. The author brings various arguments in favor of this belief and against the statement made quite frequently that Prowazek's bodies are only saprophytes.

The author believes that there are definite biologic differences between Chlamydozoon trachomatis and Chlamydozoon genitale. The relation between elementary and initial bodies is not known. The greatest theoretical difficulty at this time is that no positive culture of Chlamydozoa has ever been reported, although they become more numerous and seem to grow on the trachomatous conjunctiva. The author suggests inoculation experiments on monkeys with "ultrafiltrates" of trachomatous material.

P. C. KRONFELD.

Tumors

SURGICAL TREATMENT OF SUBCUTANEOUS ANGIOMAS OF THE FACE. T. BARDANZELLU, Arch. di ottal. **39:** 520 (Nov.-Dec.) 1932.

The author discusses the problem of treating a large subcutaneous angioma of the cheek and prelacrimal region, with a prolongation into the lower lid and caruncle. In the case reported he considered the lesions too deep for treatment with cardon dioxide snow, radium or x-rays and too large for complete removal without disfiguring scars. Through an incision at the lower orbital margin four catgut sutures were looped around the vascular tumor and a fifth was passed about the tumor of the caruncle. The skin was sutured over these catgut sutures. The cosmetic result was good, the tumors shrinking and almost completely disappearing. The remainder of the tissue was tied off later by sutures passed through the intact skin and around the tumor and cut at the point of entrance.

S. R. GIFFORD.

LEPROMA OF THE CONJUNCTIVA. M. UCHIDA, Acta Jap. Ophth. Soc. **36:** 154 (Dec.) 1932.

In a leper colony the author found four cases of leprosy of the conjunctiva. The leproma begins on the conjunctiva of the eyelid, usually near the lashes, in the middle line. The color is yellow and white. The progress is slow. Examintion showed it to contain lepra cells, fibroblasts and plasma cells, many newly formed blood vessels, lymphocytes and leukocytes, and the lepra bacillus.

A. KNAPP.

Uvea

THE CLINICAL VALUE OF HETEROCHROMIA OF THE IRIS. F. TERRIEN, Arch. d'opht. **49:** 545 (Sept.) 1932.

Whatever the mechanism of heterochromia may be, Terrien points out that the facts, which are of considerable value from a biologic point of view, are also of interest to the clinician. He first emphasizes the necessity of distinguishing heterochromia of the iris from vitiligo, which consists of an absence of uveal pigment in round patches in the ciliary

portion of the iris. Heterochromia of the iris may be divided into primary, so-called, and secondary. The secondary form follows an inflammatory condition, hemorrhage or an intra-ocular foreign body as in siderosis. Primary heterochromia should be termed "so-called," for although the color changes in the iris may appear to be the sole changes, a careful examination, especially with the slit lamp, will reveal concomitant changes in the cornea, lens and vitreous. This so-called primary heterochromia of the iris the author discusses from the point of view of heredity, which he states is a constant factor in this condition. Its association with other malformations of the eye is pointed out. He describes the corneal deposits which are frequently present, opacities in the lens, hypertension and the disturbances of the cervical sympathetic. He reports good results in these cases following operation for cataract. The theories as to its pathogenesis are reviewed, especially in the relationship to disturbances of the sympathetic nerve. He believes that further research is necessary and worth while on this phase of the subject.

S. B. MARLOW.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

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LOCAL PROTEINOTHERAPY IN OPHTHALMOLOGY. DR. M. E. LOPEZ, Madrid, Spain.

Dr. Lopez treated a patient with threatening postoperative panophthalmitis with subconjunctival injections of the patient's own blood (1 cc. four times at intervals of four days). The result was excellent. The disadvantage of the method was the production of persistent edema of the lids which finally yielded promptly to subconjunctival injections of milk, which work admirably.

RECENT VIEWS OF THE STRUCTURE OF THE MAMMALIAN RETINA. DR. F. BALBUENA, Gijón, Spain.

In the extrafoveal retina the rods and cones are grouped and form photoreceptive apparatus. Each of these consists of a cone closely encircled by rods which form a sort of sheath over the entire extent of the cone. The little globules of the rods are grouped around the base of the cone. The second, intraretinal, neuron is represented by a new, bipolar variety whose outer fiber is drawn out conically to make contact with the foot of the cone. It was not possible to get a clear interpretation of the inner fiber. Each cone unites with a single bipolar cell. From this anatomicohistologic arrangement one is entitled to conclude that the bipolar cell receives the light stimulus from the rods and cones and passes it on to the ganglion cells, and that, accordingly, this cell mediates the principal conduction of the light stimulus. The outer branches of Cajal's "bipolar rods" unite with the conical protrusions of the new bipolar cells in such a way that each rod comes into contact with at least two conical protrusions.

INFLUENCE OF SPHERICAL FORM OF LENS ON ABERRATION OF EYE FROM SPHERICAL FORM. DR. A. BIFFIS, Padua, Italy.

Dr. Biffis presents a study of the actual, parabolic form of the refracting surfaces of the human lens, assuming and reckoning with Gullstrand's constants. For the line of section of the anterior surface of the lens he accepts the equation $y^2 = 22x$; for the posterior surface, $y^2 = 11.16x$. A schematic eye with lenticular surfaces of parabolic

form is not free from spherical aberration, although this is present to a lesser degree than in schematic eyes in which the lenticular surfaces have a spherical form.

ANATOMY OF ZINN'S RING. DR. A. V. CORO, Barcelona, Spain.

The space in the upper, outer part of the orbit, bounded by the internal, external and superior rectus muscles, is an actual canal, or at least a fissure, and not a true ring, situated in the outer portion of the orbit, or rather in the posterior portion of the sphenoid fissure. Externally it is in relation with the dura covering the middle fossa, with the nerves from Willis' loop (ansa), with the blood vessels leaving the orbit and with the loose connective tissue filling the spaces in this region. Medially it is in relation with the optic nerve, from which it is separated at various points by orbital fat. An anterior and a posterior section of this fissure are to be distinguished. The former has the form of a U, placed horizontally with its opening directed backward, and is formed by the connection of the external rectus with the superior and inferior rectus muscles. The posterior portion is much more extensive than the anterior and forms a large canal. The interior of this canal is formed by the connection of the tendons of the aforementioned muscles, by the intracranial dura mater and its prolongation into the orbit or by periosteum. This so-called ring serves for the passage into the orbit of several nerves, which in penetrating are covered with dense fibrous sheaths whose dense interweaving gives one the impression that there is no connection toward the orbit. A large number of histologic specimens failed to show a ring as described by Zinn. This opening should be called the canal or fissure of Zinn.

MODIFIED PROCEDURE FOR SILVER IMPREGNATION OF THE RETINA AND VISUAL CENTERS. DR. F. BALBUENA, Gijón, Spain.

The formula for the retina is as follows: 1. Fixation in pyridine and alcohol by the method of Cajal (absolute alcohol, 50 cc., and pyridine, 5 cc., for one or more days). 2. Embedding in pyroxylin and cutting of sections of from 8 to 10 microns. 3. Immersion of the sections for from one to four hours in the following solution: potassium bichromate, 3 per cent, 15 cc.; osmic acid, 10 per cent, 2 cc., and neutral distilled (twice) glycerin, 5 cc. 4. Taking up of the sections with a glass rod and removal of the surplus of reagent fluid with filter paper. 5. Immersion in silver nitrate solution 1:2,000, with 3 or 4 drops of pyridine to 10 cc. of the solution, and heating until steam forms once or twice. The sections take on a light tobacco color. 6. Cooling and addition of a few drops of alcoholic tincture of succinic acid or of an alcoholic solution of bleached shellac. 7. Addition of a few drops of hydrochinone solution (10 per cent) to the bath. The sections darken. 8. Washing with distilled water, turning in solution of a gold compound, and fixing in hyposulphate. The result is selective staining of cones, rods and conical dilatations of the new bipolar variety.

The formula for the visual centers is as follows:

1. Fixation in a 10 per cent solution of formaldehyde for from several days to a number of months.

2. Taking of the frozen sections from the formaldehyde solution.

3. Immersion of sections in the potassium bichromate-osmic acid glycerin solution for from one to four or more hours.

4. The remaining steps, 4 to 8, are the same as in the previous formula. The result is splendid coloring of the nerve fibers.

OPTOTYPES FOR THE DETECTION OF SIMULATED MONOCULAR AMAUROSIS AND AMBLYOPIA. DR. M. GIUJARRO, Madrid, Spain.

Dr. Giujarro has perfected the Haselbach tables by substituting for letters or words whole sentences which express different ideas to one and the other eye. The attention of the simulant under examination is preoccupied with a concept which is at variance with that which originates with the other eye. Dr. Giujarro also had sentences arranged in graduated sizes of type, quantitatively corresponding, approximately, to the Weeker optotype scale of 5 minutes in the horizontal measurement, allowing amblyopia to be detected and in a given case to be accurately registered. This series also includes several optotypes for use with illiterate patients.

GONIOSCOPY: METHOD AND RESULTS, ESPECIALLY IN GLAUCOMA. DR. M. TRONCOSO, New York.

Dr. Troncoso described a new instrument, consisting of a combination of a microscope and a periscope with brilliant electric illumination, allowing the filtration angle to be examined all the way around the limbus under a linear magnification of from 13 to 27. If there is a coloboma of the iris, congenital or operative, the ciliary body and orbiculus ciliaris can be observed as far back as the ora serrata. The patient is examined lying down, and in cases of glaucoma Dr. Troncoso combines this procedure with tonometry. The results are registered graphically for later reference. A new contact glass is described which Koeppe worked out while in the United States at the suggestion of Dr. Troncoso. It is more convex than the usual model and has no depression in the upper part. It was useful mainly for making examination of the angle of the anterior chamber easier. The cases of glaucoma are arranged in a table which shows clearly that increased intra-ocular tension, even of high degree, may be present with a completely open filtration angle. In acute glaucoma the angle is open at first but gradually closes if the attacks continue and high tension persists. In simple glaucoma the angle is always open, but tends to close at one or more points if hypertension supervenes and tends to become chronic, or at least lasts some time. In glaucoma secondary to uveitis the angle is open at first but may close in part or wholly if the inflammatory process continues. It is advisable to examine the eye with the gonioscope from time to time as a routine in cases of simple glaucoma to determine the degree of patency of the filtration angle in order to determine whether

there is any immediate danger for the eye, especially in case of very high tension or marked lowering of central vision. The influence of iridectomy on peripheral synechiae, tension and vision is studied with reference to gonioscopy before and after operation.

RESEARCH ON ACTION CURRENTS IN THE RETINA. DR. L. CASCIO, Padua, Italy.

Dr. Cascio presents studies with two Siemens electrocardiographs of different models. Light excites an electric reaction in the retina of the living rabbit, which has the following peculiarities: After a short period of latency there is, although not invariably, a positive oscillation of weak intensity and very short duration. There follows a marked negative oscillation. After that, either the intensity of the current rises, reaching values above those noted in the unstimulated, quiescent state, and with a primary positive oscillation, or it remains below or at least does not exceed the intensity during the rest state. In the great majority of cases primary positive oscillations are noted, but positive secondary oscillations are exceptional. If the duration of illumination does not exceed a certain measure, an after-oscillation, always positive in character, immediately follows its interruption. The electric reactions of the retina to light are more intense after a period of dark adaptation. Local as well as general anesthesia increases the duration of the latent period and inhibits the appearance of "posthumous" oscillations. The latent period is from 0.02 to 0.1 second; the negative oscillation, from 0.1 to 0.5 millivolt; the positive primary oscillation, from 0.1 to 0.4 millivolt, and the after-oscillation, from 0.6 to 0.25 millivolt. Experiments with human eyes are not yet concluded.

HISTOLOGIC AND COMPARATIVE RESEARCH ON THE CONJUNCTIVITIS OF ERYTHEMA MULTIFORME. DR. C. PASCHEFF, Sofia, Bulgaria.

Dr. Pascheff has had many opportunities to study this condition. The eruptions on the bulbar conjunctiva appear in the form of sharply pointed flecks, nodules or papules which at times bear small white spots with a scab. Three periods are to be distinguished, one of exudation, one of papule formation and one of tissue repair. In some cases there was decided evidence of latent tuberculosis. The structure of the conjunctival eruption shows that exudative erythema multiforme is not a simple exudative process, or sort of edema of the skin, as postulated by Unna and Jadassohn, but that it is, if only temporarily, also a necrosing and productive process.

EXTRACTION OF COMPLICATED CATARACT. DR. H. ARRUGA, Barcelona, Spain.

1. Cataract complicated by tuberculosis of the uveal tract develops frequently in the persistent chronic cases and is generally accompanied by synechiae. 2. Extraction is not difficult, as the synechiae are not tough and the capsule forceps can be manipulated with ease under the iris and the delicate adhesions. 3. The prognosis is favorable, in general, and depends largely on the condition of the fundus, but as

projection tests give no definite indications in these cases one is dependent for information on examination and determination of the condition of the vitreous before operation and of the intra-ocular tension. 4. Complication with retinal detachment occurs in all cases, especially in those in which a cure has not been obtained. Extraction is simple, but one must guard against losing vitreous as this would greatly predispose to relapse of the detachment. The prognosis is generally grave, especially in young subjects.

KERATOPLASTY: EXPERIMENTAL STUDIES OF RABBITS WITH LEUKOMA CORNEAE. DR. B. CASTROVIEJO, New York.

Dr. Castroviejo reports his new method of transplanting the cornea. By beveling the edges of the corneal incision and using a knife with double, parallel blades the contact surfaces are made larger and the cavity is made exactly the same size as the transplant. The transplant is nourished and kept viable by conjunctival flaps which cover and lightly compress it. Dr. Castroviejo reports the results of forty transplantations, in 95 per cent of which there was definite healing, in 35 per cent with transparency and in 44.5 per cent with transparency at first, but only temporary and gradually disappearing. In animals with leukoma produced by lime burn the results were equally good.

DISCUSSION

DR. HILDESHEIMER: Did the transplants become vascularized?

DR. LEOZ: I had admirable results with total keratoplasty in a series of rabbits. For biologic reasons, heteroplasty will never be of any avail. One must use autoplasty or at least homoplasty and, accordingly, get a donor who is in the family.

DR. CASTROVIEJO: Occasionally the transplant became vascularized and opaque. In the cases of total keratoplasty and in those with heterotransplants it invariably became opaque. Homotransplants in a large percentage of cases remain transparent and histologically intact and are not influenced by the host.

POSSIBILITY OF CURE OF NYSTAGMUS BY (LOCAL) SURGICAL PROCEDURE. DR. N. BLATT, Bucharest, Rumania.

Dr. Blatt noted that the amplitude of the nystagmus became markedly less after an operation for squint performed on a child. Thereupon he experimented with tenotomy as a routine treatment in nystagmus, the operative section of both internal recti being followed after a lapse of from two to three weeks by tenotomy of both external recti. Transitory exophthalmos and diplopia were noted. Three patients were operated on with good results. The procedure is indicated only in purely horizontal nystagmus with "homonymous" clonic contractions, especially in idiopathic cases, which are usually congenital. The tenotomies weaken the central impulses for reflex motion, and the loss of the four recti, besides, greatly limits motion itself. The dangers and difficulties in this operation are no greater than with tenotomy in any other location.

DISCUSSION

DR. J. OHM: Operative treatment of congenital and occupational nystagmus, either by tenotomy or by advancement, has been tried frequently. Hewetson severed all four recti and was disagreeably surprised to find that the nystagmus persisted. There was nothing remarkable in this, as nystagmus is not a disease of the ocular muscles but a disturbance of innervation. It persists even in the empty orbit after enucleation, and I obtained good nystagmus curves and graphs in subjects with artificial eyes. Horizontal nystagmus could be eliminated if one succeeded in severing the nerves of both the internal and the external rectus muscles. The horizontal ocular motions would then have to be replaced by motions of the head. Probably no one would make up one's mind to perform such an operation, although it is to be assumed, or at least expected, that vision would generally be improved by this procedure. In not a few cases a good result can be attained more simply, as, for example, when the nystagmus disappears with binocular fixation to the right and gradually increases as the gaze is directed to the left. In such a case one might perform a recession operation on both dextrorotators or tenotomize both levorotators and so, perhaps, succeed in decreasing or even completely neutralizing the nystagmus with gaze directly forward. If, however, the advancement fails to produce exactly the same effect in both eyes, squint must inevitably result. Even in cases of squint combined with nystagmus, which formed the starting point for Blatt's studies, conditions may be favorable. For example, if the right eye has a convergent squint and the neutral or rest position of the left eye corresponds with the position of adduction, tenotomy of the left internal rectus will have a favorable effect not only on the strabismus but on the nystagmus as well as, and to the extent that, the position of equilibrium is shifted more to the median line. But in most cases of congenital nystagmus the eyes are quieter when in the median line than when in abduction or adduction or in lateral positions, so that in these cases recession or advancement of individual muscles is purposeless. Before attempting to attack nystagmus by way of ocular operation one must have a definite conception and a thorough understanding of the relation of this incoordination to the various positions and shifts of the ocular axes. The prognosis for cure or improvement by operation is relatively favorable in only a small fraction of the cases at best.

CAUSAL OPERATIVE MANAGEMENT OF GLAUCOMA. DR. R. SONDERMANN, Berlin, Germany.

Previous investigations by Dr. Sondermann have shown that the first consideration in treating the causes of increased intra-ocular tension is the provision of a free off-flow of blood from the eye, especially in cases in which medical therapy is indicated. Until the present time, surgical therapy has been directed exclusively toward improving the function of the equalizing valve represented by Schlemm's canal, which is, after all, of only secondary significance. Good results are probably to be explained by neoformation of venous communications between the uveal and the episcleral vascular areas. To bring about the develop-

ment of this factor must be the principal aim of causal operative treatment. Personal experience and tests have convinced Dr. Sondermann that excellent results can be attained in a field in which, so far, the usual operative therapy has not succeeded.

OLD OPHTHALMOLOGIC PRESCRIPTIONS. DR. E. PAPARCONE, Florence, Italy.

Excavations undertaken by the Italian Archaeological Mission in Egypt during the winter of 1930-1931 were successful in discovering the temple of the god Sekneptynis and nearby, among other objects of interest, a number of papyri. In one of these, reproduced and presented by Dr. Paparcone, there are two prescriptions relating to ocular disease, of which the Greek originals and their translation into Italian are presented.

SUBOCCIPITAL PNEUMO-ENCEPHALITIS AND RETROBULBAR NEURITIS. DR. M. RENEDO, Madrid, Spain.

Dr. Renedo reports the favorable effect of cisternal puncture with subsequent insufflation of air in retrobulbar neuritis.

This article will appear in full in the *Klinische Monatsblätter für Augenheilkunde.*

DISCUSSION

DR. VIDAUR agrees with Renedo and heartily recommends cisternal puncture.

DR. SABBADINI notes that previous investigations have been made by di Marzio and Reverdino. The condition in the first case reported suggests chronic hydrocephalus with chiasmal compression. It is not the insufflation of air but the relief of tension by decompression which effects the cure. In Italy only the complications of posterior sinusitis, those of multiple sclerosis and very exceptionally amblyopia due to tobacco or alcohol are considered as retrobulbar neuritis.

LOCALIZATION OF LID SYMPTOMS PATHOGNOMONIC OF EXOPHTHALMIC GOITER. DR. A. FEIGENBAUM, Jerusalem, Palestine.

Velhagen, Jr., noted in cases of encephalitis ocular symptoms which were looked on as characteristic of exophthalmic goiter, and he raised the question as to whether this proved that the ocular symptoms in this disease originated in the region of the midbrain. In two suitable cases, Dr. Feigenbaum was able to demonstrate histologically, for the first time, that these symptoms occur in association with, and as a result of, undoubted lesions on the floor or in the neighborhood of the fossa of Sylvius. In a child 7½ months of age there was noted marked internal hydrocephalus with extreme dilatation of the lateral ventricles, particularly of the third ventricle, which was distended to the size of a pigeon's egg. The cause was congenital obstruction of the aquaeductus Sylvii. A bit of tissue from the region of the third ventricle showed gliosis with proliferations. Gliosis with perivascular infiltrations was found in a second case on histologic examination of tissue from the third ventricle. It seems beyond question that the disturbance expressed

in the ocular symptoms is due partly to pressure and partly to toxic action of the cerebrospinal fluid on the gray matter of the ventricles and on the sympathetic centers which have been demonstrated there. Dr. Feigenbaum was able to demonstrate these ocular symptoms, not only in encephalitis, but in severe typhus with cerebral manifestations, in a child with osteoporosis and defects in the skull and in a number of cases of infantile or juvenile hydrocephalus. The four cardinal lid symptoms of exophthalmic goiter can appear in other diseases. They are merely different aspects of the same disturbance, rigidity of the levator palpebrae muscles. The histologico-anatomic localization of this symptom is almost surely the floor of the third ventricle or the region of the aquaeductus Sylvii.

DISCUSSION

DR. ABRAMOVICZ has observed the symptoms of exophthalmic goiter in children with serous meningitis and rigidity of almost all the muscles. This disease, as is well known, localizes itself around the ventricles, a further proof of the localization of lid symptoms in this region.

DR. LAUBER asks about the position of these eyes during sleep, and whether Bell's phenomenon was normal or inverted.

DR. VELHAGEN, JR.: Feigenbaum's observations and those of Abramovicz fill the gap which was formerly presented by the fact that in so-called thyrotoxic encephalopathy the expected changes in the central nervous system failed to materialize, or at least could not be found in the few investigations of most recent date directed to this point. These show that the lid symptoms are not especially pathognomonic and are not actual criteria of exophthalmic goiter but rather a set of focal symptoms, which, it is true, frequently appear in this disease, indicating that the central nervous system is affected in many if not all cases of exophthalmic goiter.

DR. FEIGENBAUM: Nothing can be said as to Bell's phenomenon in these cases, as the children kept their eyes wide open. I believe that the exposure of white sclera above the cornea in hydrocephalus is to be explained by the effect of this condition on the centers mentioned.

HISTOPHARMACOLOGY OF INFLAMMATION. DR. K. VELHAGEN, JR., Halle, Germany.

Dr. Velhagen induced a mild inflammation in rabbits' eyes by puncture of the anterior chamber, irritation of the iris or discission of the lens, and then made comparative quantitative tests of the original aqueous and of the anterior chamber refill for their content in histamine-like substances which would register on the isolated uterus of guinea-pigs. He found a definite increase in the speculum or titer of these substances as a result of the traumatism, and it is possible that they play a rôle in intra-ocular inflammation by virtue of their vasodilating action. As they are present in the normal lens (Braun) and are found, after discission, in increased quantity in the aqueous, they may well have some importance in certain irritative conditions following extracapsular extraction.

A Case of Epithelioma of the Bulbar Conjunctiva. Dr. J. N. Dugan and Dr. Nanavati, Bombay, India.

Dr. Dugan and Dr. Nanavati give a short report on and description of a malignant growth of the conjunctiva.

Etiology of Horner's Symptom Complex and of Heterochromia. Dr. A. Passow, Munich, Germany.

This article appeared in full in the *Archiv für Augenheilkunde* (107: 1, 1933).

"Meft." Dr. Varela Santos, Santiago, Spain.

Dr. Santos describes under this title an apparatus for the determination of refraction by color contrast. It can also be used to test visual acuity, light sense and color vision, e. g., in railway employees, or to try out colors as to their suitability for use in art and industry.

A New Slit Lamp. Dr. Comberg, Berlin.

Dr. Comberg describes an original model with the following improvements: increased economy of space by doing away with the long arm bearing the slit lamp; easy change from one examined eye to the other by a vertical arrangement of the slit lamp and lateral progression of the lamp in front of the tipped up (and back) microscope; simplification of the course of examination by vertical and horizontal displacement of the living object (eye of the patient) and its progressive movement in front of the motionless and invariably centered and focused coupled observation and illumination apparatus, giving the possibility of making a scale of definite angular values between the direction of illumination and that of observation; improvement of illumination by increase in intensity; lengthening of the slit used in ordinary examination and the possibility of a transition, at will, from slit to whole field illumination.

Treatment of Corneal Tuberculosis with Guaiacol. Dr. N. Pines, London, England.

Dr. Pines found guaiacol to be an especially useful agent in ocular tuberculosis when injected under the conjunctiva at intervals of from three to seven days.

New Keratometer. Dr. H. Hartinger, Jena, Germany.

Determination of the curvature of the cornea with the usual keratometers comes down, in the last analysis, to a longitudinal measurement of the corneal images reflected from illuminated test objects situated at known distances from each other. In order accurately to measure such mirrored objects which move with the eye, they have usually been doubled by various optical contrivances so that their images could be adjusted and superimposed. This adjustment or registering was effected either by suitably changing the position of the illuminated test marks or by altering the relations of the optical divisions of the observation

apparatus. The new keratometer constructed by the Zeiss works in Jena does away with these mechanical displacements completely and thus eliminates at the same time the sources of error connected with them. For each principal section two entirely dissimilar test marks are provided, viz., one indicator and one double division, which are imaged at infinite distance by means of collimator lenses. By means of an adjustable ocular the double division allows one to read off directly either the radius of curvature of the cornea in millimeters or the refraction value of the corneal system in diopters. If the cornea is markedly deformed by (irregular) astigmatism and the indicators and double divisions are not in the axis of the principal sections, the indicators appear displaced vertically at right angles to the divisions. By turning or rotating the observation tube and with it the collimator lenses, both principal sections can be registered simultaneously and their curvatures determined at once by a glance into the ocular. The four apertures between the reversing lenses form the diaphragm openings of the optical appliance and are projected by the reflected rays on the cornea, or at least into its immediate neighborhood, to be measured. The distance between the four entrance "pupils" are so chosen that they are within the optically active effective zone of the cornea. In this way one is assured that the curvature of the cornea is determined at exactly the same definite point or area for each fixing eye.

GENESIS OF MYOPIA. DR. G. LEVINSOHN, Berlin, Germany.

Dr. Levinsohn presents statistics of refraction tests (154) on apes. Spontaneous myopia occurred in 16 per cent, but in 63 per cent of those treated by Dr. Levinsohn's procedure. Drawings are presented to demonstrate that the anatomicohistologic basis of the myopic conus or crescent can be produced unexceptionably by his experimental method and that this tissue change corresponds almost completely to the changes in the myopic human eye. Dr. Levinsohn describes a model showing the effect of gravity on the globe when directed downward and inward when the head or the trunk is flexed. The globe is represented by a rather large rubber balloon with a double wall, and the optic nerve, by a rather firm tube entering somewhat to the nasal side of the posterior pole; the vessels proceeding from the nerve head are drawn in curving serpentine lines on the inner surface of the ballon. The anterior section of the balloon debouches into a large opening representing the cornea, which can be closed off and hermetically sealed by a glass window. The balloon is fastened with its firm "optic nerve" tube to a wooden wall in such a way that the tube exerts a pull medially from within outward. If the balloon is now filled with water and adjusted vertically or, while distended and blown up, is drawn forward and inward, the following changes can be noted where the "optic nerve" tube enters the "globe": 1. Seen from the rear without, the point of entrance moves uniformly forward and inward. 2. Seen from the glass window, the membrane adjacent to the inner wall of the tube remains behind—appears, in fact, to be drawn backward. 3. The nasal, anterior margin of the "nerve" tube becomes narrower; i. e., the nasal margin of the optic nerve becomes less distinct. 4. The temporal margin of the tube becomes

broader; i. e., the anterior temporal margin of the optic nerve, especially the lower half, becomes more sharply defined. 5. The sinuous lines representing the retinal vessels which proceed, outward (above?) and below, over the anterior, temporal margin of the tube end appear more stretched and rectilinear.

EXPERIMENTAL STUDIES OF THE VITREOUS GEL AND OF INTRA-OCULAR TENSION. DR. MARIN COHEN, DR. H. J. NEW and DR. J. KILIAN, New York.

It seems possible that some of the factors which influence the edema of the vitreous in vitro and the relation of its pressure to its volume in situ (vivo) may be endogenous and produced by vitreous metabolism. The reactions of the carbohydrate metabolism may be oxidizing or glycolytic. In order to obtain an index for oxidizing reactions the oxygen consumption of a number of animal species was determined with the aid of Warburg's microspirometer under various conditions, with and without the addition of grape sugar. The relation of volume to pressure in the vitreous of animals was studied in vitro by noting the diffusion of solutions of various electrolytic and nonelectrolytic substances in the vitreous gel. The total osmotic pressure fraction corresponding to the colloids was determined in the vitreous of animals. Proteins separate out from the vitreous when filtered under pressure. The effect of increasing the protein content of the vitreous on vitreous edema in vitro and on the intra-ocular vitreous in situ was determined by the introduction of known quantities of protein, separated by filtration, into the vitreous cavity. Diminution of the colloid osmotic pressure of the blood serum took place when animals were placed on a protein-low diet. The authors also studied the influence on intra-ocular tension of this alteration in the colloid osmotic tension as a result of lack of protein and of the consequent fall in colloid tension. They give a description of experimental methods which will ensure an increase in intra-ocular tension in animal eyes. They discuss the theories of the etiology of glaucoma.

ENCEPHALOMYELITIS OR MULTIPLE SCLEROSIS? DR. J. KLAR, Kattowitz, Poland.

Dr. Klar reported, in 1932, a series of cases, the number of which has subsequently increased, under the title of "Acute Optic Encephalo-Myelitis." The symptoms are those of acute retrobulbar neuritis, generally with papillitis or choked disk and amaurosis of several weeks' duration. In abortive cases there is amblyopia, often with involvement of the optic tract. Other "nervous" disturbances are not essential or important. Generally pyramidal symptoms (Rossolimo) are present; more infrequently, severe myelitis, slight changes in the cerebrospinal fluid, slight pleocytosis, with a positive globulin and colloid test and the appearance of grape sugar in the fluid. There is no increase worth mentioning in the cell content. Inoculation of cerebrospinal fluid into the cornea of rabbits had positive results (keratitis punctata superficialis of Knauer and Jaensch, whose results with multiple sclerosis were negative).

(To be continued)

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

ALGERNON B. REESE, M.D., *Secretary*

May 15, 1933

MARK J. SCHOENBERG, M.D., *Chairman*

A COSMETIC LENS FOR PERSONS WITH MYOPIA OF HIGH DEGREE.

DR. HAROLD G. NOYES and MR. THEODORE E. OBRIG, A.B.

The flat lenticular lens, introduced forty years ago, was almost as thick and ugly as the ordinary double concave lens. Because of the spherical aberration produced around the edge of the concave disk in polishing the surrounding plano band, the disk had to be ground to about 30 mm. in diameter. A disk of 30 mm. produces a much thicker lens than does a disk of 20 mm.

In 1903, Carl Schneider invented and patented the well known convex edge lenticular lens. This was an improvement on the flat lenticular lens in two ways: First, it produced a lighter lens with the same size disk. Second, it did away with most of the spherical aberration, because although the disk was ground in the same manner as for the flat lenticular lens the surrounding band of glass was ground and polished strongly convex with a deep curved tool, rather than with a flat one which caused the polishing cloth to come in contact with the finished disk.

The authors presented a new and modern form of the flat lenticular lens, free from any distortion or spherical aberration in the concave disk. The method of manufacture has never been used before. With this method, a smaller disk can be used, the weight of the lens reduced one-third, and the cosmetic effect of the lens greatly increased. A disk of 20 mm. is recommended for lenses of 17 diopters or over, and a disk of 25 mm. for corrections under 17 diopters. These sizes will give the patient a field large enough for general wear. The cylindric correction is ground on the outside surface of the lens and is accurately centered with the optical center of the concave disk. These lenses are now being made exclusively by Gall & Lembke, Inc., of New York City, under the trade name of Myo-Disc. The price is the same as that of the old lenticular type.

DISCUSSION

DR. MARK J. SCHOENBERG: Are these lenses much more expensive than the ordinary ones?

DR. NOYES: The cost, as I understand it, is about $3 more per pair than for the double concave lenses worn by the average person with a high degree of myopia.

THE PROPER TIME FOR OPERATION IN STRABISMUS.

DR. JOHN H. DUNNINGTON.

This article was published in full in the October, 1933, issue of the ARCHIVES, page 438.

RETRACTION SYNDROME. DR. RUDOLF AEBLI.

This article was published in full in the November, 1933, issue of the ARCHIVES, page 602.

THE PROLONGED OCCLUSION TEST, WITH ILLUSTRATIVE CASES. DR. WENDELL L. HUGHES.

This article will be published in full in a later issue of the ARCHIVES.

INDICATIONS FOR TREATMENT FOR COMBINED LATERAL AND VERTICAL STRABISMUS. DR. JAMES W. WHITE.

This article was published in full in the November, 1933, issue of the ARCHIVES, page 585.

THE INTIMATE RELATION BETWEEN OPHTHALMOLOGY AND NEUROLOGY: SOME REMINISCENCES. DR. BERNARD SACHS.

This article was published in full in the October, 1933, issue of the ARCHIVES, page 433.

Book Reviews

The Main Afferent Fiber Systems of the Cerebral Cortex in Primates. An Investigation of the Central Portions of the Somato-Sensory, Auditory, and Visual Paths of the Cerebral Cortex, with Consideration of Their Normal and Pathological Function, Based on Experiments with Monkeys. By Stephen Poliak, M.D., Associate Professor of Neurology, Department of Medicine, University of Chicago. University of California Publications in Anatomy. Volume 2. Price, $10. Pp. 370, with 96 figures. Berkeley, Calif.: University of California, 1932.

This fine monograph is a preliminary, and a necessary one, to a study of the comparatively little known association connections of various cortical areas as determined by cyto-architectural and myelo-architectural investigations.

To obtain results applicable to human physiology and pathology, the experiments were carried out on monkeys (Macacus rhesus, except in one case in which a Java monkey was used). The experiments consisted of interruptions of the afferent projection fiber systems at or near their origin from the diencephalon, thereby causing their partial or complete secondary degeneration, avoiding at the same time as far as possible other fiber systems. The secondarily degenerated fibers were traced to their respective terminations in the cortex, and the internal arrangements of the three afferent paths investigated (somatosensory, auditory and visual), the cortical areas in which they terminated and the finer relation of their fibers to the cortex were studied. The staining method used was the osmic method of Marchi, which displays the fibers, in a certain stage of degeneration, stained black against a light field.

Experiments were also made with a view to ascertaining, in sections stained by Nissl's method, the cells in the external geniculate body from which various portions of the visual radiation originate. This method takes advantage of the well known changes manifested in nerve cell bodies the axons of which have been cut, known as "primary irritation of cells" or "retrograde degeneration." In this case lesions of various parts of the visual cortex were made and the resulting changes in the cells of the external geniculate body studied.

Conclusions regarding the three afferent paths investigated were obtained from eleven experiments, brief mention being made of twenty-five other experiments.

Regarding the somatic sensory system, and without entering into the comparison made by the author of the areas receiving degenerated fibers with the cyto-architectural areas determined by Brodmann in Cercopithecus, the interesting result was obtained that the somatosensory cortical area included a large precentral as well as a postcentral area, thus disagreeing with the view probably at present most widely accepted, i. e., that the somatosensory area is wholly postcentral. On the other hand, by comparing the somatosensory area with the auditory and visual areas as determined by the same research, it is evident that considerable

areas separate these and are therefore apparently destitute of afferent projection fibers, thus approaching the view of Flechsig that large cortical areas are solely "association" areas. There are no gaps or islets in the supply of thalamic fibers to the area. The intracortical somatosensory fibers are somewhat coarser than those of the visual radiation. None cross in the corpus callosum. Comparisons are made by the author with the cyto-architectural areas of Brodmann and other investigators.

The author discusses the possible functions of the somatosensory area. He does not regard the entire area as a functionally uniform "sensomotorium," as he believes that this view is negatived by the various structural differences within it; but he thinks that his investigations "clearly demonstrate that some form or other of the receptive, somatosensory function has to be attributed also to the precentral agranular cortex."

The cortical area supplied by the auditory radiation is the upper half of the superior temporal convolution, and only that. Only a few fibers reach the free surface of the temporal lobe.

The author concludes from the spatial arrangement of the cochlear fibers that there is a spatially arranged system for the conduction of various tones and has put forward an interesting attempt to explain the minute function of the auditory system based on the arrangement of the direct and spiral cochlear nerve fibers.

Of the three systems, the visual receives the most attention. Comparisons of the effects of more extensive and partial lesions appear to show clearly the spatial arrangement stressed by the author in his summary, which is given later. Quite remarkable is the strict correspondence of the distribution of the visual fibers with the area striata, and it is also interesting to note the confirmation of the views of Ramón y Cajal, based on Golgi preparations, as to the layers receiving the exogenous visual fibers, i. e., the various sublayers of the stripe of Gennari.

The following is the summary, with some minor omissions, by the author of the results deduced from his detailed description of the visual system:

"(1) The external geniculate body of the between-brain must be recognized at present as the only and the exclusive origin of the visual radiation in primates.

"(2) There exists only one single direct afferent visual path from the subcortical region to the cerebral cortex. (Nothing positive is known of indirect visual paths.) It is the fiber system originating from the external geniculate body. That external geniculo-cortical radiation forms a strong fiber lamina or layer identical with the 'primäre Sehstrahlung' of Flechsig, with the stratum sagittale laterale of H. Sachs, a portion of which was called by Burdach, Wernicke, and Monakow 'fasciculus longitudinalis inferior' of the parieto-occipital and temporal lobes. However, other fibers: efferent, callosal, and associational, mix with the afferent visual fibers. Stratum sagittale internum, 'sekundäre Sehstrahlung' of Flechsig, is an efferent fiber system. The tapetum is composed exclusively of callosal fibers.

"(3) The cortical region where the visual path terminates is a single, definite, and sharply delimited area which is identical with the area striata of G. E. Smith, field 17 of Brodmann, or area OC of Economo-Koskinas distinguished by the strong intracortical fiber layer, the stripe of Gennari or Vicq d'Azyr. No other areas of the cerebral cortex receive afferent visual fibers.

"(4) The central visual path above the external geniculate body is strictly unilateral. No evidence exists of a partial decussation of its fibers through the corpus callosum ('fasciculus corporis callosi cruciatus'). The only spot, therefore, where the visual path undergoes a partial decussation is below the between-brain in the optic chiasm.

"(6) The visual radiation is composed of fibers grouped into fiber bundles arranged, on the whole, in parallel fashion. Each fiber bundle originates in a definite small segment of the external geniculate body and terminates in a definite small segment of the striate area.

"(7) The visual radiation or the external sagittal layer of the parieto-occipital lobe can be subdivided into three anatomical-functional portions discernible on cross-sections perpendicular to the long axis of the hemisphere: (*a*) the dorsal horizontal branch, (*b*) the ventral horizontal branch, and (*c*) the vertical or the perpendicular branch connecting both horizontal branches. The dorsal horizontal branch is composed of fibers originating in primates from the medial (internal) segment of the external geniculate body close to the thalamus which corresponds with the upper extramacular segment of both homonymous hemiretinae, including the upper monocular portion of the crossed retina. This branch supplies the striate cortex of the upper (dorsal) lip of the fissura calcarina. . . . The ventral horizontal branch is composed of fibers originating from the lateral (external) segment of the external geniculate body close to the lentiform nucelus, corresponding with the lower extramacular quadrants of both homonymous hemiretinae, including the lower monocular portion of the crossed retina. It supplies the striate cortex of the lower (ventral) lip of the fissura calcarina. . . . The perpendicular (intermediate) branch of the visual radiation is composed of fibers originating from the large intermediate segment of the external geniculate body, which is situated between both internal and external segments of that body. This intermediate segment corresponds to both dorsal and ventral quadrants of both homonymous hemimaculae. The vertical branch represents a considerable portion, probably more than half, of the visual radiation. It supplies the striate cortex covering the pole of the occipital lobe, and in the monkey, as far as the striate cortex extends, the external face of the occipital lobe or the so-called operculum occipitale. The vertical branch represents in its upper half, near the dorsal horizontal branch, the upper quadrants of both homonymous hemimaculae; in its ventral half, near the ventral horizontal branch, the lower quadrants of both homonymous hemimaculae. . . .

"(9) The bilateral or the double cortical representation of each total macula, with each and all of its receptor elements connected with both hemispheres, has no anatomical foundation. The individual macular elements are represented only in one hemisphere. But each half of each

macula is projected upon another hemisphere. In other words, each macular cortex represent homonymous halves of both maculae.

"(10) The entire striate area receives everywhere afferent visual fibers. There exist no visible gaps (small cortical zones without an afferent fiber supply) which might separate small supplied cortical islets. The number of afferent visual fibers per cortical square unit appears to be larger in the occipital pole and in the occipital operculum than in the calcarine fissure (in the monkey). The coarse exogenous fibers of the visual cortex entering more or less obliquely the lower strata and terminating in the zone of the stripe of Gennari or Vicq d'Azyr, described by Ramón y Cajal, are afferent visual fibers originating in the external geniculate body of the same hemisphere.

"(11) The small segments of the visual cortex supplied by individual bundles of the visual radiation have sharp limits and definite (triangular) form. . . . Each triangle of the visual cortex gets its own bundle independently from the neighboring triangle. There does not, accordingly, exist an overlapping of these small zones, a mingling of their respective bundles, or even a 'diffuse' termination of individual bundles of the visual radiation in extensive portions of the visual cortex. Neighboring triangles are supplied by neighboring fascicles of the visual radiation.

"(12) The entire afferent visual system from its beginning in the peripheral organ (retina) to its termination in the cerebral cortex (striate area) is composed of individual receptor and conductor units. These structural and functional units are strictly arranged according to the 'spatial principle' on the one hand, and according to the 'principle of neighborhood' on the other. Each area striata is in some sense a faithful copy of both homonymous halves of both retinae. There exists a full preservation of 'spatial relations' in the visual cortex as it exists in the retina, though the absolute shape of the 'cortical hemiretina' is somewhat changed in consequence of the slight mutual displacement of neighboring units or segments.

"(13) All visual impulses whatever their special form or quality may be, and regardless of their ultimate destination, so far as they reach the cortex of the forebrain, first go to the striate area from which they are distributed to other regions of the same and the opposite hemisphere.

"(14) Since there exists a fixed arrangement of functionally different bundles or segments of the visual radiation (present experiments) as well as of the peripheral portion of the visual system (Rönne, Brouwer-Zeeman), which means a fixed projection of definite retinal quadrants upon definite portions of the visual cortex, symmetrical in both hemispheres, the injuries of the visual system will produce definite symptoms depending on the location and the extent of the injury. Since each of the constituent structures of the afferent visual system and of the visual cortex has its own function, its destruction will be followed by an irreparable and lasting loss of that particular function. The replacement or compensation of the lost function must be achieved by what can be called a more economic utilization of the parts remaining undamaged."

Combining the results of his investigations with those of Brouwer-Zeeman and others, the author has furnished an interesting figure showing the positions of the quadrants of the visual fields, the macular,

perimacular and monocular areas being distinguished from each other in the retina, the external geniculate bodies and the visual cortex. The author concludes that "no evidence and no necessity exist for a bilateral cortical representation of each macula in the sense of Wilbrand, Lenz *et al.* The preservation of macular vision must be explained by the (1) separate course and protected position of the macular portion of the visual radiation and a well protected location of the human macular cortex, and (2) by the close position of those portions of the striate area which represent extramacular portions of the visual fields, in both lips and in the bottom of the fissura calcarina, which arrangement renders separate injury to those latter regions possible (beside the multiple blood supply of the occipital pole)."

The author furnishes an interesting summarization of various possibilities from unilateral injuries or pathologic processes which are limited to the occipitoparietal and temporal lobes which cannot, for lack of space, be given here.

It is certainly to be hoped that Dr. Poliak will be able to carry out his difficult program so well begun. Even this portion might be amplified and strengthened by further experimental work along the present lines of research.

OLIVER S. STRONG.

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INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem.: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.

Ophthalmological Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay, 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., W. 1, London.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Time: 1934.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. William C. Finnoff, 1612 Tremont St., Denver.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: 1934.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Ophthalmological Society

President: Dr. W. G. M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Lucerne, Que. Time: July 9-11, 1934.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. W. Morse, 66 W. Park St., Butte, Montana.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President, Dr. A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Place: New Orleans. Time: 1934.

Southwestern Michigan Triological Society

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 920 Republic Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Edward J. Whalen, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. W. R. Bedingfield, 753 Broad St., Augusta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.
Place: Augusta. Time: May, 1934.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Peter T. Grant, 148 Monroe Ave., Grand Rapids.
Secretary: Dr. Ralph B. Fast, 1410 Bank of Kalamazoo Bldg., Kalamazoo.
Place: Battle Creek. Time: 1934.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Gordon B. New, Mayo Clinic, Rochester.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. S. T. Hubbard, 294 State St., Hackensack.
Secretary: Dr. G. S. Laird, 125 Central Ave., Westfield.
Place: Atlantic City. Time: June, 1934.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.
Secretary: Dr. Webb W. Weeks, 20 E. 53rd St., New York.
Place: Utica. Time: May 14-16, 1934.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. O. Ruud, 318 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Valley City. Time: 1934.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Robert C. O'Neil, 105 Waterman St., Providence.
Secretary: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. O. S. Hodges, 388 Pearl St., Beaumont.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. R. Slopanskey, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.
Secretary: Dr. Edwin W. Burton, 104 E. Market St., Charlottesville.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.
Secretary: Dr. W. F. Beckner, 1050 5th Ave., Huntington.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Browne Morgan, 260 Liberty St., Bloomfield.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Colin Campbell, 170 St. George St., Toronto.
Secretary: Dr. H. M. Macrae, 170 St. George St., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Benjamin Rones, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Justin M. Waugh, Cleveland Clinic, Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. P. Chamberlain, 7405 Detroit Ave., Cleveland.
Secretary: Dr. A. D. Ruedemann, Cleveland Clinic, Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. T. S. Love, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Daniel Brannin, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Raleigh R. Snyder, 410, 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President, Dr. Edward B. O'Keefe, 901 State St., Schenectady.
Secretary-Treasurer: Dr. W. C. Mott, 118 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Valin R. Woodward, Flatiron Bldg., Forth Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. D. A. Bartley, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. M. B. Simpson, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Clifford S. Losey, 219 E. 10th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Los Angeles County Medical Association, 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Karl N. Victor, Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. George B. Trible, 1801 I St., Washington.
Secretary: Dr. Harry F. Davies, 1835 I St., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Frederick E. Hasty, 119, 7th Ave. N., Nashville, Tenn.
Secretary-Treasurer: Dr. Kate Savage Zerfass, 803 Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Webb William Weeks, 20 E. 53rd St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Abbott M. Dean, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: Second Tuesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. W. E. Munroe, 277 Alexander St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Max W. Jacobs, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert S. Irvine, 490 Post St., San Francsico.
Secretary: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey, Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. J. L. Walter, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. H. O. Brust, 820 S. Crouse Ave., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelmann, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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